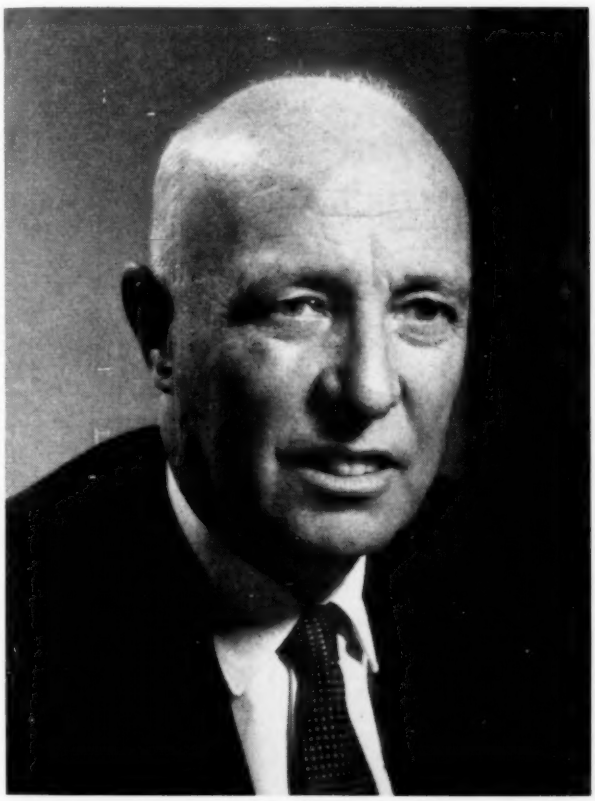
Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

Kaapstad, 10 Junie 1961 Deel 35 No 23 Volume 35 Cape Town, 10 June 1961

DR. H. S. GEAR

NEWLY APPOINTED SECRETARY-GENERAL, WORLD MEDICAL ASSOCIATION

The appointment of Dr. Harry Gear to be Secretary-General of the World Medical Association, with its headquarters in New York, brings yet another South African into greater prominence in the international sphere. It is a source of great satisfaction to his many colleagues that Dr. Gear should be the man singled out of the hundreds of thousands of doctors in the world for this high honour and important post.



Dr. Gear

Dr. Harry Gear is the eldest of three brothers who are all rendering distinguished service in medicine. His career commenced with distinction as a student when, after obtaining an Honours B.Sc. degree, he was awarded the Association's Medal as the 'Most distinguished graduate' in the M.B., Ch.B. class of his year (1928).

He married Joyce, the daughter of Sir William Leishman, whose name is notable in medical science, and their three sons all show signs of ability in the careers of service which they have chosen. It may not be generally known that, after post-graduate study in public health and tropical medicine in London, Dr. Gear spent some years in China as head of the Department of Preventive Medicine at the Lister Institute for Medical Research in Shanghai. He then returned to South Africa where he joined the Public Health Department and rose to the position of Deputy Chief Health Officer in the Cape. During this time he served on the Federal Council and took a keen interest in the affairs of the Medical Association of South Africa. During the war years Dr. Gear served with distinction in Africa and the Middle East, attaining the rank of Lieut.-Colonel and being mentioned in dispatches.

Dr. Gear represented the Union Government at the founding of the World Health Organization in 1946, and was its representative at four World Health Assemblies. In 1951 he was seconded from the Department of Health to become Assistant Director-General of the World Health Organization in Geneva. His work took him to many countries as consultant on health matters, and he occupied this position until 1958.

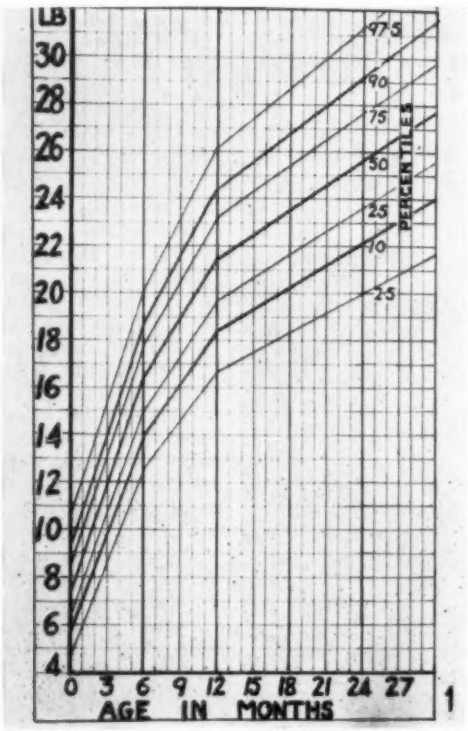
In the following year he served as a member of the Governor-General's Commision of Enquiry into the medical services of the Federation of Rhodesia and Nyasaland, and also travelled in North America and Western Europe as a Fellow of the Rockefeller Foundation. Last year he succeeded General Orenstein as Director of the Pneumoconiosis Bureau of the CSIR.

With the growing prominence of African Affairs, the Council of the World Medical Association has chosen well in appointing this quiet, unassuming, internationally-minded man as its Secretary-General. His wide knowledge of medical affairs in the world, and especially his knowledge of Africa, will be of great value to the Organization.

Dr. Harry Gear will leave his mark on the World Medical Association and all that it stands for, and the Association will be the better for it.

WEIGHT CHARTS OF COLOURED INFANTS AND PRE-SCHOOL CHILDREN

ISOBEL ROBERTSON, B.A., M.B., CH.B., D.P.H., *Maternal and Child Welfare Officer, City Health Department, Cape Town*



1

Weight charts have been prepared from the known weights of normal Coloured infants and pre-school children seen at child-welfare centres, crêches, and nursery schools in Cape Town.

To ensure that these charts represent the normal range of weights for Coloured children, 400 children were chosen, all of whom were in good health and from families with incomes above the poverty datum line. This excludes malnourished children.

Each child's weight was recorded at successive ages. From these weights percentiles were calculated and charts prepared (Figs. 1 and 2). The charts are presented as a series of percentile lines, the 50 percentile representing the mean, and the 2·5 and 97·5 percentile lines the very uncommon limits of the normal. Incidentally, these limits are exactly twice the standard deviation on either side of the mean.

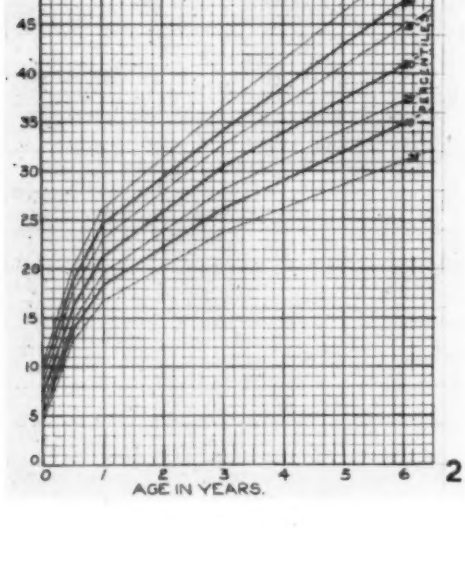
The percentile chart published by the Children's Medical Center, Boston,* has been reduced to the same scale for comparison. The rate of growth shown on the Boston chart is somewhat greater than that shown for Coloured children (Fig. 3).

An example of how the charts can be used to demonstrate the state of nutrition and rate of growth is shown in Fig. 4.

It is submitted that these percentile charts should be used as a standard for assessing the rate of growth and nutritional state of Coloured infants and pre-school children.

* Anthropometric Weight Chart—The Children's Medical Center, Boston.

Fig. 1. Percentile weight chart for Cape Coloured infants.
Fig. 2. Percentile weight chart for Cape Coloured children from birth to 6 years.
Fig. 3. Percentile weight chart for American children from birth to 6 years.



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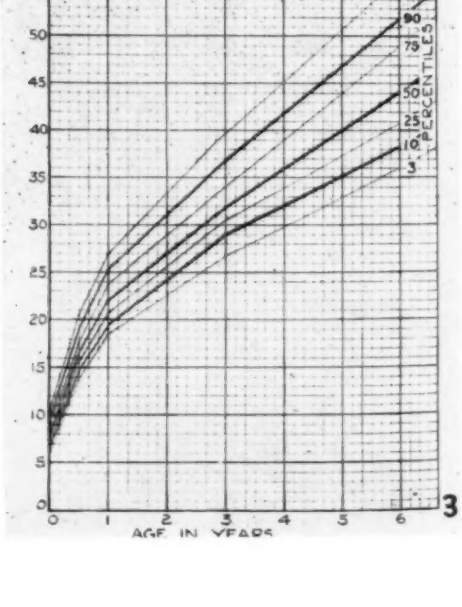

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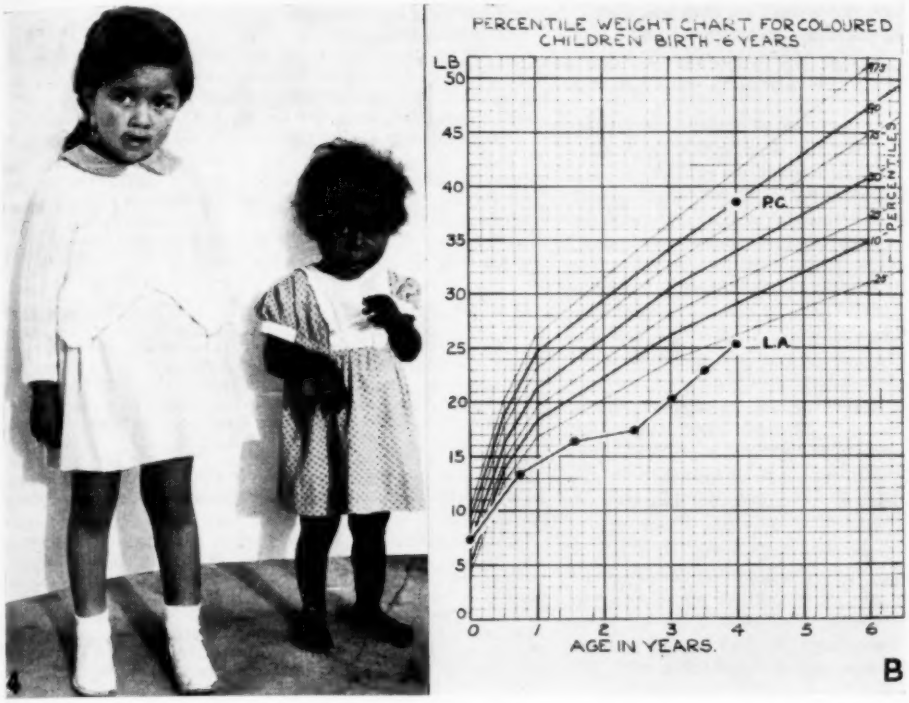

Fig. 4. Example of the use of the percentile chart. *A.* P.C. (left) and L.A. (right) are both 4 years of age. *B.* P.C.'s weight has been plotted on the percentile chart, and falls on the 90 percentile line, i.e. well above average weight. L.A., on account of severe malnutrition from early infancy to 2½ years, reflects weight well outside the 2·5 percentile line, i.e. severely underweight. From the age of 2½ years L.A. has had milk added to her diet, and has shown a marked increase in the rate of gain in weight for the last 1½ years, which is clearly shown on the chart. At this rate of growth L.A. will soon regain normal weight and may even approach P.C.

ASPEKTE VAN POSTPARTUM KOLLABERING IN PRE-EKLAMPSIE EN EKLAMPSIE

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Dit is bekend dat sommige pasiënte wat pre-eklampsie of eklampsie het, partykeer skielik in die onmiddellike puerperium kollabeer sonder enige trouma of bloeding om so 'n skokbeeld te verklaar. Sonder onmiddellike behandeling is baie van die gevalle noodlottig.

Oor die laaste 30 jaar het daar verskeie artikels verskyn oor pasiënte wat skielik dood is, óf voor óf na kraam, waar by outopsie gevind is dat daar hewige bynierbloedings was. Dit was toe algemeen aanvaar dat die bynierbloeding verantwoordelik was vir die kliniese beeld. Oor die laaste dekade is hierdie bewering egter in twyfel getrek. Symington[1] en Shearman[2] het toe voorgestel dat daar 'n kliniese entiteit van akute byniertekort, sonder enige bloeding in die bynierweefsel, is.

Gevalle is gerapporteer wat nie net gedurende swangerskap of na kraam voorkom nie, maar ook na enige operasie, bloeding, of sepsis.[2-5]

Daar is twee groepe waarin die gevalle voorkom:

1. Voorafgaande byniertekort soos Addison se siekte, of waar die bynier onderdruk is deur die toediening van die kortin groep hormone.

2. Vorige normale persone wat skielik akute byniertekort ontwikkel. Dié beeld kom meer dikwels in pre-eklampsie en eklampsie voor as in enige ander toestand.

Patologie

By outopsie is daar baie min veranderinge te vind. Shearman[2] sê: ‚A most impressive clinical picture, leaves very little evidence with present autopsy methods, of the metabolic storm that kills the patient'. In gevalle waar daar 'n chroniese tekort is, is die byniere kleiner as normaal as gevolg van atrofie van die zona fasciculata; die medulla is nie aangetas nie.[6] In vorige normale gevalle is die histopatologie heel anders—daar is vergroting van die korteks met veelvoudige trombose wat ook die medulla partykeer raak. In erge gevalle is die bynier net 'n sak bloed. Dit moet egter onthou word dat die bynier-bevindings as gevolg van die skok mag voorkom en nie 'n oorsaak is nie.

Dit is tot nou toe beweer dat die byniere gedurende swangerskap hipertrofeer. Hierdie bewering word nou egter in twyfel getrek. Daar word gevoel dat dié bewering nog nooit bevestig is nie, en dat die bynier eintlik gedurende swangerskap onderdruk word deur die kortikosteroïde van die plasenta. Daar is dus dieselfde resultaat as wanneer kortisoon toegedien is.[7]

Dit is bewys dat daar gedurende die laaste trimester 'n verhoogde kortikoïde vlak in die bloed is, en dat dit veral verhoog is in gevalle van pre-eklampsie en eklampsie.[8,9] Daar word oor die algemeen aangeneem dat hierdie produksie die gevolg is van plasentale aktiwiteit. Hills[10] beweer egter dat daar moontlik lewer-patologie kan wees sodat detoksikasie van die 17-hidrokortikosteroïde nie normaal uitgevoer word nie.

Die proses van kraam verwyder nou die plasenta en dus die bron van die hormone. Daar is dus 'n relatiewe tekort aan die kortisoon groep van hormone, want die bynier wat nog geatrofeerd is, kan nie betyds kortisone vervaardig nie

(halflewe van hidrokortisoon is 3-5 uur). As daar nog daarby pre-eklampsie is, is die algemene vatkrampvernouing wat voorkom verantwoordelik vir nog meer atrofie van die bynier, en dus is die sloering, voor vervaardiging hervat word, nog langer.

As die drukte* nou te groot is, dit wil sê in spontane kraam, keisersnit of tangverlossing, dan kan daar 'n skielike byniertekort wees met kollabering en dood van die pasiënt.

Daarby moet nog onthou word dat die steroïde wat opgaar in die weefsels, edeem met soutretensie veroorsaak, veral die aldosterone wat 20-30 keer meer natrium terughou as D.C.A. en 500 keer meer as hidrokortisoon. Alhoewel die totale hoeveelheid liggaamsout verhoog is, is die serum-natrium verlaag, terwyl die kalium verhoog is. By gewone behandeling van gevalle van pre-eklampasie word 'n beperkte natrium inname toegelaat, en dus is daar nog 'n laer serum-natrium. Tatum en Mulé[11] het bewys dat serum-natrium lesings van 110 mEkw./l. gevind is in 16 pasiënte wat gekollabeer het na kraam, keisersnit, of tangverlossing. Ten spyte van hidrokortisoon het die bloeddruk in dié gevalle nie eintlik baie gestyg nie, of as dit gestyg het, was dit maar tydelik. Die toediening van natrium-kloried, 0·9 G. per 100 ml., het 'n dramatiese styging van bloeddruk meegebring!

Kliniese Beeld van Akute Byniertekort

Die pasiënt wat voorheen 'n verhoogde bloeddruk gehad het, het nou skielik geen pols of bloeddruk nie. Ten spyte hiervan lyk sy heeltemal redelik. Die vel is warm en sy is heeltemal helder psigies. Later begin sy sianoties te raak en nog later raak sy koud en begin dan deurmekaar te raak. Sy sterf gewoonlik binne 5-8 uur tensy sy behandel word. Daar is altyd 'n eosinofilie.

As aan hierdie pasiënte noradrenalien gegee word, sal die bloeddruk tydelik styg, maar later ontwikkel daar 'n weerstand en meer en meer noradrenalien is dan nodig om die bloeddruk te bestendig. Later help dit ook nie meer nie. As hidrokortisoon nou gegee word, sal noradrenalien weer die bloeddruk laat styg. Die rede hiervoor is dat die kortikosteroïdes nodig is vir die sensitiwiteit van die kapillêre vate vir noradrenalien.

Vogt[12] verklaar: ‚The corticoids bestow on the tissues the capacity to react efficiently to internal and environmental stimuli, but are not in themselves those stimuli. The loss of reactivity of at least some tissues to noradrenalin would fit that conception'.

In teenstelling met die gewone bloedvat-reaksie by skok, dit wil sê vatvernouing, is daar dus voorafgaande vatverwyding met 'n stadige perifere bloedsomloop.[13] Die vel is dus warm en sianoties.

BEHANDELING

1. *Profilakse*

'n Eosinofieltelling van meer as 600 per ml. dui 'n byniertekort aan.

Dit is raadsaam om hidrokortisoon, 100 mg. per 1,000 ml. 5% dektrose in water, te gee in gevalle van erge pre-eklampsie of eklampsie gedurende en na kraam, of veral met chirurgiese ingrepe soos, byvoorbeeld, keisersnit of tangverlossing.

2. *Aktief*

(*a*) 100 mg. hidrokortisoon moet onmiddellik gegee word. Dit kan herhaal word na 3-4 uur as dit nodig is. As die reaksie gunstig is, kan 25 mg. hidrokortisoon per mond of per inspuiting gegee word. Dit word stadig verminder tot niks oor 5 dae.

*Drukte - stress

(*b*) Vervang bloedverlies waar nodig.

(*c*) Noradrenalien.

(*d*) Dit moet onthou word dat baie van die pasiënte te veel vog kry en dan pulmonêre edeem ontwikkel. Wees dus versigtig vir té ywerige toediening van vog.

(*e*) Natrium-kloried, 0·9 G. per 1,000 ml., binne-aars.

In die Afdeling Verloskunde by die Karl Bremer-hospitaal, Bellville, is dit 'n roetine-gebruik om by elke geval van eklampsie of erge pre-eklampsie wat 'n chirurgiese ingreep kry, 100 mg. hidrokortisoon deur indruppeling toe te dien.[14]

Tot datum was daar net 2 uit 141 gevalle van erge pre-eklampsie en eklampsie wat gekollabeer het. Ek wil graag hierdie twee gevalle kortliks bespreek.

Geval 1

A.B., Kleurlingvrou, 26 jaar, primigravida. Toegelaat as 'n noodgeval met 'n geskiedenis van antepartum eklampsie. Met ondersoek was die fundus 36 weke, die fetale hart hoorbaar, en sy was nie in kraam nie. Soos die gebruik by dié hospitaal is, is 'n keisersnit op haar uitgevoer. Daar is met indruppeling van 5% dekstrose in water begin, maar as gevolg van 'n misverstand is geen hidrokortisoon in die bottel geplaas nie. Teen die einde van die operasie het die pasiënt se bloeddruk skielik gedaal van 200/160 mm.Hg tot 50/0 mm. Hg. Op hierdie stadium is dit toe gevind dat geen hidrokortisoon in die indruppeling was nie, en 100 mg. solukortef is onmiddellik binne-aars gegee. Die bloeddruk het dramaties gestyg.

Geval 2

X.Y., Kleurlingvrou, 32 jaar, para 7. Toegelaat as 'n noodgeval met 'n geskiedenis van intrapartum eklampsie. Sy het geboorte geskenk aan 'n dooie baba voor toelating. By toelating het die pasiënt 'n warm vel gehad. Die naels en lippe was sianoties. Haar bloeddruk was 40/0 mm.Hg en die pols 92 per minuut. Sy het wakker gelyk en het haar geskiedenis helder gegee. Daar was geen pyn nie en bloedverlies was min. Die uterus was goed saamgetrek. Daar was geen urine-uitskeiding nie.

Een ampule noradrenalien is met 100 ml. 5% dekstrose in water-indruppeling gegee. Die bloeddruk het gestyg tot 110/40 mm. Hg maar na 'n ruk het dit weer gedaal na 50/0 mm. Hg. Nog 2 ampules noradrenalien is bygevoeg in die bottel en weer was daar 'n tydelike styging van die bloeddruk. Op hierdie stadium is 100 mg. hidrokortisoon bygevoeg in die indruppeling en die bloeddruk het begin styg tot by 180/90 mm. Hg; die indruppeling moes toe baie stadig gestel word. Die pasiënt het na 2 uur 'n bloeddruk van 110/70 mm. Hg behou sonder noradrenalien.

Hierdie twee gevalle is bespreek om die waarde van hidrokortisoon te beklemtoon in gevalle van erge pre-eklampsie of eklampsie.

SUMMARY

1. Some aspects of postpartum collapse in cases of severe pre-eclampsia and eclampsia are discussed.
2. The importance of acute adreno-cortical deficiency is stressed.
3. The possibility of sodium-chloride deficiency is discussed.
4. Treatment with hydrocortisone is discussed.

Ek wil dr. R. L. M. Kotze, Mediese Superintendent Karl Bremer-hospitaal graag bedank vir toegang tot die rekords.

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Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE NAVORSER IN SUID-AFRIKA

'. . . He lived by measuring things
And died like a recurring decimal
Run off the page, refusing to be curtailed;
Died as they say in harness, still believing
In science, reason, progress . . .
'. . . His mind developed like an ancient church
By the accretion of side-aisles and the enlarging lights
Till all the walls are windows and the sky
Comes in . . .'

Louis MacNeice

Gedurende die jongste tye het daar uit alle oorde waarskuwings gekom dat ons in hierdie land op 'n gevaarlike lae peil staan ten opsigte van navorsers en navorsingsgeleenthede. So het dr. H. O. Mönnig, voorsitter van die Adviesraad vir Wetenskaplike Beleid, byvoorbeeld, onlangs gesê dat net sowat drieduisend van Suid-Afrika se driemiljoen blankes, nl. 0·1 persent, met navorsing besig is. En van hierdie groep is heelparty net deeltyds daarmee besig, soos in die nywerhede, die universiteite, ens.

Hierdie syfer is veels te laag as 'n mens die land se totale bevolking in ag neem. Volgens beskikbare gegewens is daar in Amerika waarskynlik 6 of 7 keer meer navorsers (op 'n proporsionele en vergelykbare basis beskou) as wat daar in ons land is. As ons op hierdie grondslag voortgaan, sal dit nie net vir ons moeilik wees om tred te hou met die menigvuldige moderne ontwikkelinge op haas alle gebiede van die lewe nie, maar ook sal dit die onvermydelike agterstand waarmee elke jong land begin, groter laat word in plaas van kleiner.

Die oorwegings wat ons so pas genoem het, geld ook op die gebied van mediese navorsing. Aan die begin wil ons dit duidelik stel dat ons dankbaar is vir die groot ondersteuning wat navorsers en navorsingsinrigtings, wat aan ons universiteite verbonde is, van sulke instansies soos die Suid-Afrikaanse Raad vir Wetenskaplike en Nywerheidsnavorsing ontvang. Dit het ons in staat gestel om die kerne van hoogstaande navorsingseenhede, wat werk van briljante gehalte doen, aan ons universiteite op te rig. Met die oog op toekoms en die bepaling van ons plek in die mediese wêreld, moet ons egter 'n veel meer verbeeldingryke en omvattende plan bedink om navorsingsgeriewe daar te stel.

Mediese navorsing word in Suid-Afrika op die oomblik op verskillende vlakke benader.

1. In die eerste instansie is daar die individuele navorser wat gewoonlik ook in die aktiewe mediese praktyk self staan. Dit sou ons in die verleentheid bring om hier name te noem, maar almal van ons is bekend met daardie onvermoeide yweraars, wat as enkelinge versprei is oor ons land, wat letterlik op hul geestelike en liggaamlike kapitaal leef omdat die navorsingswerk wat hulle so onvermoeid doen, eintlik as oortydwerk beskou moet word. Wat ons op hierdie gebied nodig het, is die oprigting van groot en sterk finansiële stigtings wat dit moontlik sal maak vir navorsers, wat bewys het dat hulle uitstaande talent het, maar nie genoegsame finansiële bronne om hul projekte bevredigend te ontgin nie, om hul werk met toegewyde ywer voort te sit, sonder om hulle (terwyl hulle met hul navorsing besig is) te bekommer ,oor die dag van môre en oor wat hulle sal eet of drink'. Met al die krag van ons oortuiging wil ons hier 'n beroep doen op ons eie Suid-Afrikaanse Rockefellers en Carnegies om te voorskyn te kom. Sommige nywerheidsleiers het al reeds betreklike groot somme geld beskikbaar gestel vir navorsingswerk aan ons universiteite en vir die navorsing van spesifieke mediese probleme. Maar die groot Suid-Afrikaanse navorsingstigting, wat onafhanklik staan van enige groepsbelang en wat as uitsluitende oogmerk het die bevordering van suiwer en toegepaste navorsing, wag nog op sy prins.

2. Sedert die stigting van die Suid-Afrikaanse Instituut vir Mediese Navorsing in 1912, het hierdie inrigting 'n belangrike rol gespeel op die gebied van navorsing in ons land. Die doelstellings van die Instituut kan soos volg saamgevat word: (*a*) Navorsing na die oorsake en metodes van beheer van menslike siektes; (*b*) laboratoriumondersoeke van 'n diagnostiese aard vir gesondheidsoutoriteite, hospitale, die mynbedryf, en andere; en (*c*) die voorbereiding van biologiese produkte vir die voorkoming en behandeling van siektes. Die navorsers wat aan die Instituut werk, doen dus werk van suiwer en toegepaste aard, en die meeste van hulle werk in voltydse hoedanighede. Werksomstandighede en geleenthede vir navorsing moet egter so aantreklik gemaak word dat die Instituut, wat reeds 'n lang en trotse rekord het, kan voortgaan om van die beste talent onder ons mediese wetenskaplikes te trek.

3. Mediese navorsing het ook geweldige vooruitgang gemaak as gevolg van die navorsing wat aan ons universiteite gedoen word. Hierdie navorsing word ondersteun en is tot 'n groot hoogte moontlik gemaak deur die ondersteuning wat ontvang word van die Suid-Afrikaanse Raad vir Wetenskaplike en Nywerheidsnavorsing wat na die laaste oorlog opgerig is. Ook word die navorsingsbedrywighede aan ons mediese skole moontlik gemaak deur die stelsel van gesamentlike mediese dienste, waarvolgens die universiteite en die provinsiale departemente op 'n vennootskapsgrondslag verantwoordelik is vir die personeeluitrusting van onderwyshospitale.

Dat daar uitstekende navorsingseenhede aan al ons mediese skole verbonde is, ly geen twyfel nie. Nogtans wil ons dit hier stel dat die vereistes van opleiding aan die een kant, en suiwere navorsing aan die ander kant, tog nog verbrokkelend werk. Daar moet veel meer, as wat op die oomblik die geval is, voorsiening gemaak word vir voltydse navorsingsbetrekkings aan ons universiteite. Ons moet die beste talent waaroor ons beskik vir die werk trek, en ons moet die werkers ooreenkomstig hul bydrae uitstekend beloon. Dit gebeur nog veels te veel dat toegepaste nywerheidsorganisasies beter besoldiging aanbied as wat aan ons universiteite beskikbaar is, sodat ons ons basiese navorsers verloor.

4. Groot farmaseutiese firmas speel ook al meer en meer 'n belangrike rol in die navorsing na siektes en middels. In die verlede was daar dikwels die neiging vir die mediesgeskoolde navorser en sy kollega in die farmaseutiese nywerheid om mekaar se bydraes te misken. In hierdie opsigte het dinge nou radikaal verander. Daar is nou egter baie meer fasette verbonde aan mediese navorsing en diens as ooit tevore. As ons die nodige brûe kan bou, behoort die mediese en farmaseutiese professies saam te staan in 'n kragtige front teen onkunde en siekte.

Die wetenskaplike navorser in die algemeen, en, sover as wat dit ons aangaan, die mediese wetenskaplike navorser in die besonder, in watter hoedanigheid hy ook al werk, beklee 'n sleutelposisie in ons ontwikkelende beskawing. Om die beste belegging vir die toekoms te maak, moet ons ons navorsers versigtig uitsoek, hulle heelhartig aanmoedig en ondersteun, en dit vir hulle moontlik maak om hul bydraes op die hoogste vlak te lewer.

CONTROL OF POTENTIALLY HARMFUL DRUGS

It had been proposed by the Minister of Health to introduce legislation amending Section 65*bis* of the Medical, Dental and Pharmacy Act during the present session of Parliament. However, agreement could not be reached between the various interested parties. The Medical Association and the Pharmaceutical Society of South Africa are opposed to the proposed amendments, and in favour of amendments put forward by the South African Medical and Dental Council and the South African Pharmacy Board.

It has therefore been decided *not* to proceed with the proposed amendments until agreement can be reached. The Secretary for Health has informed the Association that, pending the introduction of suitable amendments, it is the intention of the Minister to relax administratively the application of the less important provisions of Section 65*bis*. Accordingly, no action will be taken in the meantime in respect of minor technical infringements made in good faith. The Secretary for Health points out, however, that prosecutions will still be instituted in the case of deliberate infringements.

We appeal to all our members to observe the spirit of the law, and remind them that, while minor technical infringements will not be penalized, persons who attempt to flout the requirements of the law or deliberately infringe the provisions of Section 65*bis* will still lay themselves open to prosecution.

In the *Journal* for 26 November 1960 we published an editorial[1] detailing the provisions of Section 65*bis* and appealing to doctors to put their house in order regarding prescriptions they write for potentially harmful drugs. At that time pharmacists had made the point that many doctors were contravening various provisions of this Section of the Act, and, while it may be that matters have improved in the intervening months, it may be of value to set forth the provisions of the Section once again as they stand at present.

Section 65*bis* lays down the requirements for prescriptions for potentially harmful drugs extremely clearly. It states that these prescriptions should include the following information: The date of issue of the prescription; the name and address of the patient; the name and quantity of the drug to be supplied, the number of times and the intervals at which the prescription should be dispensed and, except in the case of a preparation for external use, the amount and frequency of each dose to be taken; and the usual signature, address, and professional qualifications of the doctor who issued the prescription, which prescription should be in the doctor's own handwriting.

Provision is also made, in the case of an emergency, for a pharmacist to supply potentially harmful drugs on the telephonic instructions of a doctor personally known to him. A prescription in the prescribed form, covering such instructions, should then be forwarded within twenty-four hours by the doctor to the pharmacist.

Until agreement is reached and suitable amendments passed by law, Section 65*bis* still stands. As soon as such amendments have been made we shall inform the profession: until such time we appeal once again to our members to see to it that they observe the requirements of the law regarding these prescriptions.

1. Editorial (1960): S. Afr. Med. J., 34, 1009.

THE MAST CELLS AND MASTOCYTOSIS*

WITH SPECIAL REFERENCE TO BONE CHANGES

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Paul Ehrlich was the first to describe the mast cells clearly. What is more, he did this in his first scientific paper which was published in 1877 when he was still a 23-year-old student.[1] His paper contained a plate of drawings of granular cells stained with aniline dyes, and he described their metachromatic staining with dahlia violet. Two years later he gave these cells a name.[2] Influenced by the fact that they were most numerous where there was increased local nutrition as a result of chronic inflammation or lymph stasis, he believed them to be overnourished cells, and therefore called them 'mast cells'. In the same way an overfed goose is known in many parts of Europe as a 'gemästete Gans'.

How these mast cells fitted into the human economy,

* Guest lecture presented at the Congress of the Dermatological Sub-Group (M.A.S.A.), Cape Town, 17 April 1960.

however, was completely unknown, and it was not until 10 years later that Unna[3] described the accumulation of these cells in the lesions of a rare skin disease called urticaria pigmentosa or 'Nettleship's disease'. This disease was first described in 1869 by the young British doctor, Nettleship,[4] who later became an ophthalmologist. He presented the case of a 2-year-old girl with an unusual form of urticaria in which the wheals were very persistent and left a light-brown hyperpigmentation. Rubbing the lesions caused urtication.

Both this disease and the mast cell itself remained medical curiosities. Only in the last 10 or 20 years has more light been shed on these subjects. The 'riddle of the mast cell' has by no means been solved, but it is proving to be one of the most fascinating of the cells found in animal connective tissue. In fact, it seems to have been something of a dark horse; it may yet prove to be a master cell of the connective tissue.

The chief characteristic of the mast cell is the presence in its cytoplasm of granules which stain metachromatically. By this is implied that the granules change the colour of the dye used in staining them.

Recent biochemical and histochemical studies have suggested an impressive number of possible functions for the mast cell. Since these cells are present in any area of connective tissue, and since connective tissue is so widely distributed, their physiologic activity is of great interest. This is especially so now that it is known that hormones such as the steroids exert their effects largely as a result of connective-tissue response. It seems that the mast cells produce or store in their granules a number of powerful substances. These seem to be similar to or identical with heparin,[5] hyaluronic acid[6] and histamine.[7] It has been claimed with less convincing evidence that serotonin, or 5-hydroxy-tryptamine, is also contained in mast cells.[8] These agents may be released by simple extrusion of granules, new ones forming within the cell, or sometimes as a result of total disintegration of the cell.

FUNCTIONS OF MAST CELLS

The following is a list of possible functions of mast cells related to a knowledge of the actions of these various chemicals:

1. *Heparin production*
   - (*a*) Prevention of blood coagulation
   - (*b*) Local detoxification
   - (*c*) Formation of collagen
   - (*d*) Inhibition of hyaluronidase
   - (*e*) Metabolism of fats
2. *Hyaluronic-acid production*
   - (*a*) Integrity of the ground substance
3. *Histamine production*
   - (*a*) Activation of defence and repair mechanisms (?)
4. *Serotonin production* (?)

1. *Heparin Production*

(*a*) *Prevention of blood coagulation.* The mast cell has been regarded as a single-celled endocrine gland pouring heparin into the blood stream. Although there is usually no obvious bleeding tendency in urticaria pigmentosa, and blood coagulation studies are often quite normal, cases have been described in which there were petechial eruptions and large purpuric areas in and around the lesions.

A case in a child, reported by Waters and Lacson,[9] is of especial interest. The child developed cutaneous lesions of urticaria pigmentosa in the first week of life. From the age of 3 years there was a tendency to bruise easily and a difficulty in arresting haemorrhage after minor trauma. The child developed leukaemia, in which tissue mast cells appeared in the blood. Bleeding time became prolonged and the prothrombin activity was reduced. Death occurred following the rupture of a large subcapsular haematoma in the liver. At autopsy the blood in the peritoneal cavity was found to be entirely fluid, and no clot had formed.

Some experiments on hibernating animals are relevant in this respect.[10] It is known that the blood-clotting time of these animals is short during the active summer period and prolonged during winter hibernation. Bats in summer were kept under temperature conditions of 5°C. Their blood-clotting time increased, and so did the number of mast cells in the duodenum. When winter bats were exposed to a temperature of 23°C. the reverse was observed. It seemed that the alterations in clotting time were at least partly determined by the availability of mast-cell heparin.

(*b*) *Local detoxification.* The theory has been advanced that heparin or related substances may neutralize noxious amines resulting from cell death, metabolic processes, or other factors.[11] The complexes formed are then taken up by ordinary fibroblasts. This theory is based on some well-conducted experiments.

(*c*) *Formation of collagen.* It has been shown that heparin can precipitate collagen fibrils from procollagen,[12] which may be secreted by fibroblasts in the inflammatory response. However, a number of other substances besides heparin may also cause such precipitation of collagen fibrils.

(*d*) *Inhibition of hyaluronidase.* Heparin is capable of inhibiting the action of hyaluronidase. This may indicate that mast cells play a part in the defence of the connective tissue against bacterial invasion or the spread of malignant tumours.

(*e*) *Fat metabolism and atherosclerosis.* Heparin seems to be concerned in the normal transport and metabolism of fat by stimulating 'clearing factor' production. This factor breaks up the large emulsified fat droplets in the blood stream, thus facilitating the passage of neutral fat through the capillary walls.[13] It has been further shown, as a result of animal experiments and a study of human autopsy material, that subjects with marked atherosclerosis have significantly reduced mast-cell counts in the cardiac muscle and other tissues.[14] The autopsy reports so far published on patients dying from malignant mastocytosis have made no special comment on the presence or absence of atherosclerosis, even in aged persons. It might be of value if in future special attention was paid to this aspect.[15]

2. *Hyaluronic-acid Production*

The opinion that hyaluronic acid is secreted by mast cells is based on a good deal of experimental evidence. Any increase in water content of the ground substance of connective tissue seems to cause an increase in the number of mast cells, which then undergo disruption. The hyaluronic acid liberated converts the free water into a hydrated gel and a perivascular oedema is changed into a mucinous material. Thus the physicochemical integrity of the ground substance is preserved.

3. *Histamine Production*

Although histamine has well-known and powerful pharmacological actions, its place in normal physiology is not clear.

Its release from mast cells in response to trauma or other damage to the tissues might flood the areas with protein-rich oedema fluid as a result of increased vascular permeability. This might then mobilize the fixed tissue cells before the process of repair.

The urtication which may occur on rubbing the lesions of urticaria pigmentosa is most easily explained on the basis of the local liberation of a histamine-like substance. There have also been reports of more generalized disturbances in this disease attributed to release of histamine. These include attacks of generalized flushing, either spontaneously or as a result of irritation of the lesions. More severe disturbances resembling 'histamine-shock', sometimes with loss of consciousness, have also been described. In some cases such attacks ceased after excision of a large, active, mast-cell tumour.[16] More attention has been paid to this peculiar, but rare, associated phenomenon in recent years, and some cases have been described in South Africa.[17] In some of the cases the histamine content of the lesions, the blood, and the urine was much increased.

4. *Serotonin Production*

It is less certain whether serotonin (5-hydroxy-tryptamine, 5-HT) is contained in human mast cells, although it seems to be present in the mast cells of rats. This substance is present in the enterochromaffin cells of the intestinal tract and in carcinoid tumours of these cells. It is also found in blood platelets and in some cells of the nervous system. The symptoms of the release of serotonin include, among others, such vasomotor disturbances as generalized flushing. It also causes intestinal hypermotility, and in this respect it is of interest to note that abdominal pain and diarrhoea have often been found in association with urticaria pigmentosa.

However, there is no clear evidence of raised serotonin levels in either the lesions or the blood of patients with this disease, nor has there been an increase of its metabolic end-product in the urine of these patients. Further investigation is therefore necessary to decide whether serotonin, either alone or combined with histamine, plays any part in the many-faceted symptomatology of mastocytosis.

Summary of Functions

So far as present knowledge goes, mast cells may be considered as unicellular endocrine glands. They apparently secrete a number of very powerful agents—heparin, hyaluronic acid, histamine, and possibly serotonin—mainly as part of the mesenchymal reaction in time of stress. The cells respond both to local tissue changes and to alterations in the general balance of the body hormones.

URTICARIA PIGMENTOSA OR MASTOCYTOSIS

This disease, like the mast cell itself, has emerged from relative obscurity only in the last decade. In spite of earlier reports of occasional enlargement of lymph glands and rather vaguely defined blood disturbances, we were all taught that this disease was a mere dermatological curiosity with no real significance as regards the general health of the patient. Most cases still do seem to be confined to the skin and to be entirely benign, but each year further cases are reported in which systemic involvement has been detected. This systemic involvement may also be benign, but it occasionally takes a malignant course and may prove fatal. At least 12 fatal cases have been described.

The systemic involvement takes the form of mast-cell infiltrations in the lymph nodes, liver, spleen, bones, haemopoietic system, and sometimes other organs.[18] Lupus erythematosus was once also considered a purely dermatological condition; then it was found not only to have possible systemic associations, but also to exist in a purely systemic form without skin lesions. By analogy it was prophesied that one day a purely systemic form of urticaria pigmentosa would be detected. Such cases without any cutaneous involvement have now been described, e.g. a purely 'splenic mastocytosis',[19] and a condition of 'mast-cell leukaemia'.[20,21] Therefore, the use of the general term 'mastocytosis' instead of urticaria pigmentosa seems fully justified.

Recent literature contains a number of excellent short surveys on urticaria pigmentosa, and among the best of these is certainly that of Marshall *et al.*[17] These authors proposed an interesting system of classification of the mastocytoses. We, too, had been thinking on somewhat similar lines and the following is the result of such an attempt at classification:

1. Cutaneous mastocytoses

(*a*) Localized
  (*i*) Solitary mastocytoma
  (*ii*) Grouped lesions
(*b*) Disseminated (benign urticaria pigmentosa)
  (*i*) Juvenile type
  (*ii*) Adult type
(*c*) Diffuse (mastocytose diffuse cutanée)

2. Cutaneo-systemic mastocytosis

Any of the forms listed under cutaneous mastocytosis, with associated mast-cell invasion of lymph nodes, liver, spleen, bones, haemopoietic system, etc.

(*a*) Benign
(*b*) Malignant

3. Systemic mastocytosis

Involvement of the above-mentioned structures, together with the viscera, but without lesions of the skin.

It is now clear that the complete and accurate diagnosis of a case of urticaria pigmentosa or mastocytosis must be based, in addition to the dermatological examination, upon a thorough general clinical examination; a full haematologic study, including that of the coagulation mechanism; an examination of the bone marrow or histologic study of a bone biopsy specimen; and a complete radiological study of the entire skeleton.

Although there were earlier hints and suggestions that urticaria pigmentosa might in some way be a systemic disease, these have only recently been substantiated. In 1949 Ellis[22] reported on an autopsy performed on a one-year-old child who died in the active stage of the disease. During life there had been no suspicion of systemic involvement, but the postmortem examination revealed numerous mast-cell infiltrations in the bone marrow, thymus gland, liver, spleen, pancreas, and lymph nodes.

Tissue mast cells in the blood were first described by Hissard and co-workers[23] in 1950, in a case with the diffuse type of skin involvement. Although the usual number of mast cells in the circulating blood was only 1-2%, they rose to 45% following splenic puncture, which presumably induced contraction of the spleen. The patient eventually died.

Bone Changes

The existence of widespread bone changes in a patient with urticaria pigmentosa was first demonstrated by X-ray examination in 1950 in Israel.[24]

The patient was a 53-year-old man whose widespread skin lesions had appeared 4 years earlier. He was admitted to hospital for thorough general examination. An X-ray picture of the chest showed that the ribs were involved by a cystic osteoporotic process, with punched-out areas and thickening of the trabeculae. The changes resembled those seen in multiple myeloma or metastatic carcinoma.

Since the pattern of the X-ray shadows was reminiscent of that produced on the skin by the pigmented lesions, it was thought that they might be due to the same process. Sclerotic bone changes were found in the skull, the pelvis and the lumbar vertebrae. The bones of the extremities seemed unaffected. No mast cells were detected in the bone marrow and the patient refused to allow diagnostic costotomy. Follow-up X-ray studies performed over a period of 7 years revealed progression of the sclerotic process in the bones, even though the patient remained in relatively good general health.

The late Dr. Charles C. Rein of New York visited our clinic and saw this patient. On his return to the USA he re-examined all his patients with urticaria pigmentosa, and together with Dr. Clyman[25] reported a case of this disease with localized bone changes. They suggested that a central registry should be set up in Jerusalem to which reports on skeletal studies in urticaria pigmentosa might be forwarded.

As a result of cases reported to this registry,[26] and of others described in the literature, it seems that a total of well over 30 cases of this disease with osseous changes have been detected since 1952. It is not possible to be certain how many cases there have been in which X-ray studies were performed, but showed no changes, since many of these will have gone unreported. Two out of 20 cases examined in our clinic had bone changes.

The second of these 2 patients is of especial interest.[27,28] She was a 55-year-old woman with urticaria pigmentosa of the maculopapular type, of 5 years' duration. Her general health, at first, was good. Skeletal X-ray examination, which was a routine procedure since the finding of bone changes in the first patient, showed that she, too, had generalized bone changes. This patient was under observation for 2½ years. Then, her general health suddenly deteriorated and she developed a high fever and died with a clinical picture of monocytic leukaemia.

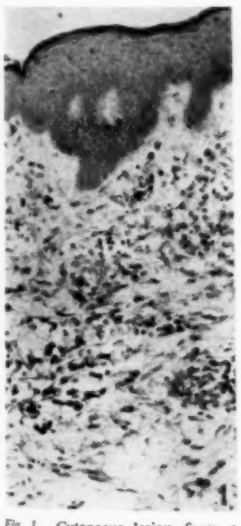

Fig. 1. Cutaneous lesion from a patient with mastocytosis. Mast cells are dispersed throughout the middle and upper dermis. One month later the patient died from monocytic leukaemia (toluidine blue × 470).

A comparison of the first X-ray pictures with those taken shortly before her death, showed that there had meanwhile been a marked progression of the sclerotic changes, both in the areas originally affected and in bones which had previously appeared normal. Thus, in the earlier pictures the margins of the inner and outer tables of the skull were distinct, and islets of increased density could be seen scattered throughout the bones. In the later X-ray pictures islets could not be seen, for the outer and inner tables now formed one continuous undifferentiated sclerotic mass. In the later pictures the previously normal-looking distal portions of the radius and ulna, and the carpal and metacarpal bones, were seen to have become involved by the same sclerotic process.

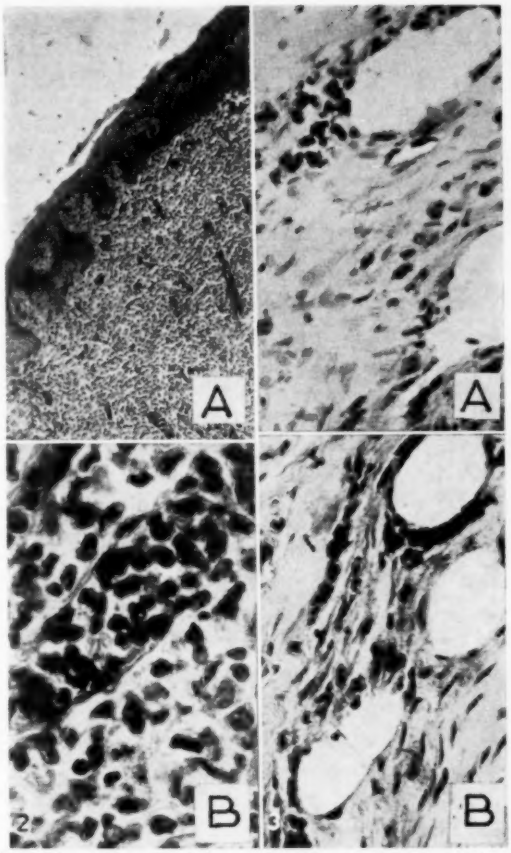

Fig. 2. Same patient as Fig. 1. Cutaneous lesion removed at autopsy. *A.* Low power. There is a dense cellular infiltration of the entire dermis (toluidine blue × 125). *B.* High dry power. The infiltrate consists of monocytes, with intermingled mast cells.
Fig. 3. Same patient as Fig. 1. Lumbar vertebra. *A* and *B.* Aggregations of tissue mast cells in the bone marrow. Mast cells are also seen lining large, empty, vascular spaces (toluidine blue × 400).

Shortly before death the patient developed a leukocytosis, rising to 248,000 white blood cells per c. mm. Seventy-eight % of the cells were monocytes, some of which were classed as 'atypical'. Autopsy studies confirmed the clinical diagnosis, and proved that the changes in the bones seen at X-ray were indeed due to mast-cell infiltrations. Many of the marrow spaces were either completely fibrosed or were filled with accumulations of mast cells. These cells were also present within large, empty, vascular spaces (Figs. 1—5).

The bone changes so far described in mastocytosis may be classified in two groups: Generalized or diffuse involvement, and localized involvement.[29]

In *generalized or diffuse involvement*, exemplified by the cases I have described, the lesions seen in the X-ray pictures are diffusely scattered throughout the affected bones. The bones most frequently affected are those of the central column of the body—i.e. the skull, vertebrae, ribs and pelvis. Nearly all the patients in which this type of involvement has been described were over 30 years of age.

In *localized involvement*, the changes are seen as localized areas, sometimes with a sharply 'punched-out' appearance,

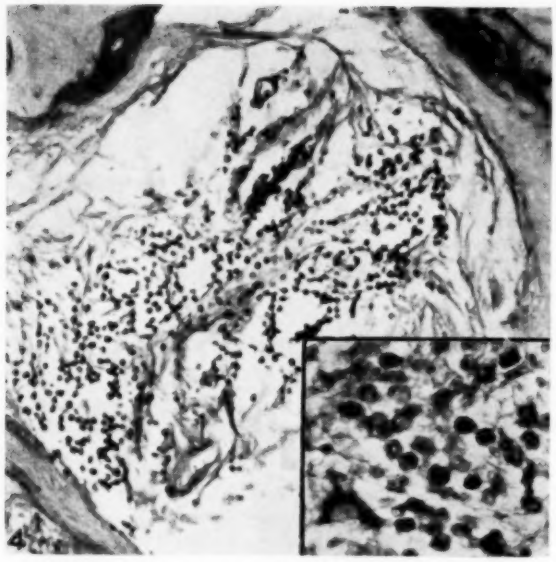

Fig. 4. Same patient as Fig. 1. Lumbar vertebra. Accumulation of mast cells in marrow space, which also shows early fibrosis (toluidine blue ×105). Inset: Typical mast cells in the marrow space (×370).

confined to one part of the affected bone. The skull and the bones of the extremities have shown such lesions, but the ribs and vertebrae are usually not involved. This type of bone lesion has been described in children as well as in adults.

Treatment

On the subject of the treatment of mastocytosis I will say little. Much has been tried, but, in general, with far from satisfactory results. Antagonists of heparin, histamine, hyaluronic acid, and serotonin have been tried, in addition to steroids, radiation therapy, and many less specifically directed procedures. The chief importance of treatment once seemed to be only cosmetic, but now that possible systemic and fatal developments must be considered, the search for effective treatment takes on a new urgency.

It seems likely that the solution will be linked to a greater understanding of the 'riddle of the mast cell'.

SUMMARY

1. The possible functions of the mast cells are discussed.
2. Their association with urticaria pigmentosa is described.
3. The disease urticaria pigmentosa, or mastocytosis, is described in detail and a tentative classification of the mastocytoses is given. The widespread nature of mastocytosis is stressed, with special reference to the bone changes and the possibility of malignancy.
4. The unsatisfactory nature of the available treatment of mastocytosis is mentioned, and the urgency of finding effective treatment is stressed.

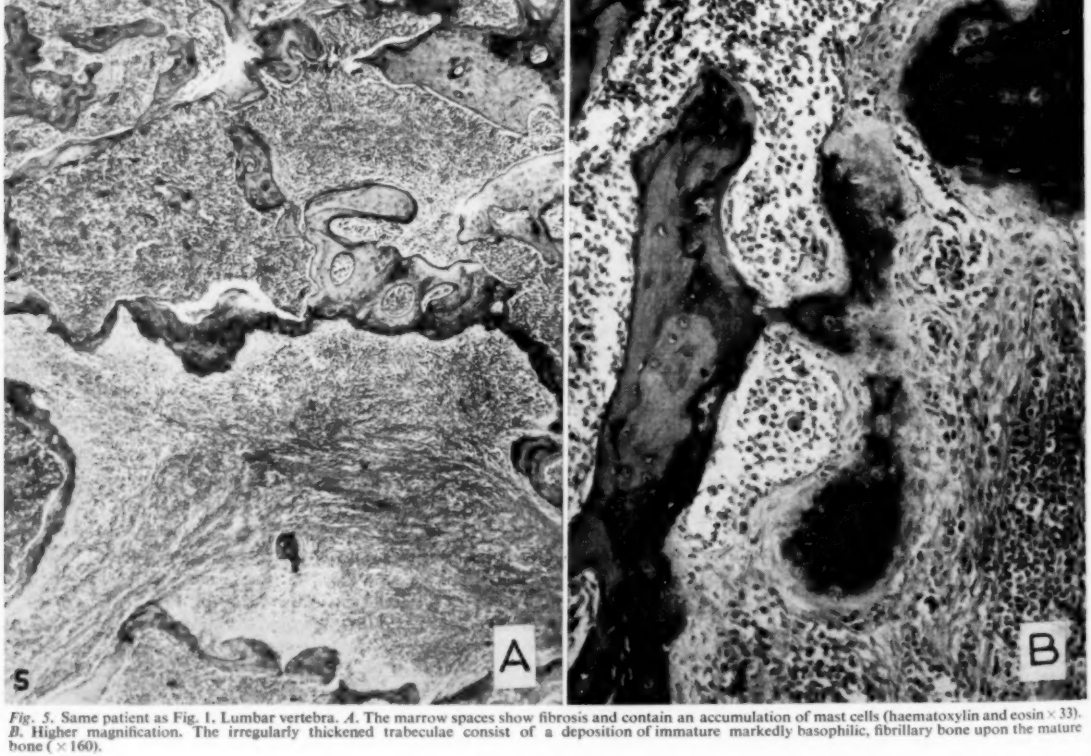

Fig. 5. Same patient as Fig. 1. Lumbar vertebra. *A*. The marrow spaces show fibrosis and contain an accumulation of mast cells (haematoxylin and eosin ×33). *B*. Higher magnification. The irregularly thickened trabeculae consist of a deposition of immature markedly basophilic, fibrillary bone upon the mature bone (×160).

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THE PRESENT-DAY STATUS OF THE CONSTRICTION RINGS OF LABOUR

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In spite of the noteworthy advances in most obstetrical fields during the past two decades, one of the subjects which still remains highly speculative is the physiology of uterine-muscle contractility. The aetiology of constriction-ring dystocia is closely linked with the physiology of uterine action, and it is for this reason that it remains obscure, with the treatment unsatisfactory. On the surface it would appear that the management of this condition is straightforward, but any complacency that exists will soon be dispelled by a glance at the appallingly high foetal and maternal mortality and morbidity rates. A review of the subject was undertaken to try to assess the correct lines of treatment when faced with this not uncommon condition.

Definition

The term constriction ring refers to an abnormality of uterine contractility during labour either due to, or responsible for, the production of a ridge of uterine muscle, which is circular or nearly so, and which projects into the uterine cavity or disturbs its normal shape.

Incidence

The estimations vary, but the incidence of this condition is probably nearer 0·1%[1] than 1%[2,3] of all labours. Primigravidae, multigravidae and grande-multiparae may all be affected.

TYPES

Rudolph[4] has divided these rings into 2 varieties:

1. *Reversible*. This is simply a spasm affecting a localized circular area of the muscle of the uterus (Fig. 1). It can develop quite quickly following intra-uterine manipulation, or the administration of oxytocic drugs in the presence of hypertonus. This is the type of ring which should in theory respond to conservative measures.

2. *Irreversible*. Here there is a thick localized ridge of uterine muscle which projects into the cavity of the uterus and grips the foetus (Fig. 2). The upper segment is thinned out from the uterine fundus towards the ring, and the lower segment usually presents the same appearance. It would seem that this type of ring usually occurs in association with abnormal uterine action and is demonstrable at Caesarean section. It persists even after death as shown by Phillips[5] and Louw.[6] Phillips regards this condition as the end-result of a colicky uterus in an extreme degree, where the spasm assumes an annular distribution.

In a review of 371 cases collected from the literature, Rudolph[4] further subdivided these constriction rings into:

A. *Those palpable internally* (91%).

1. Around the baby's neck, behind the symphysis pubis (75%).

2. Around the baby's body, above the symphysis pubis (16%).

3. In front of the presenting part, behind the symphysis pubis (9%).

B. *Those palpable externally* (9%).

Sites

(*a*) *The junction of the upper and the lower segments*. This is the commonest site, and here it is important to distinguish between a constriction ring and a Bandl's ring or retraction ring (Fig. 3). A retraction ring is the normal demarcation between the upper and lower segments that can be felt from time to time as a slight ring-like elevation in the cavity of the uterus.

(*b*) *In the upper segment*. These rings have been described as occurring most commonly around the infant's neck, waist, or limbs, but it is conceivable that they arise anywhere in the upper segment due to contraction of the circular muscle fibres over a point of slight resistance.

(*c*) *In the lower segment*. This is a rare site for the occurrence of constriction rings, and some doubt has been cast on whether they arise here at all.

It is also worth remembering that the ring may be better developed either on the left or right,[6,7] or even anteriorly or posteriorly. That more than one site may be affected simultaneously has also been shown.

AETIOLOGY

As has been pointed out, the disturbance is basically one of uterine function in labour, which results in the constriction of an area of circular muscle fibres. This may occur at various levels in the uterus.

Rudolph and Ivy[4,8] demonstrated that physiological constriction rings form in the uteri of pluriparous animals, where the foetus is contained in segments of the two uterine

horns. These horns are the counterpart of the upper uterine segment in the human. The rings, by the very nature of their function, are situated at the junction of the upper and lower uterine segments, and in the upper uterine segment. Furthermore, Ivy, Hartmann and Koff[9] have shown that the normal contraction wave can be reversed by stimulating the lower segment in the monkey. From these observations they have suggested that under certain conditions atavistic tendencies are exhibited by the uterine musculature, and that these rings are then formed at the corresponding sites in the human uterus.

Stress, resulting from inadequate or improper preparation for pregnancy and labour, may produce conditions favourable for the formation of a ring. Against this argument, however, is the well-documented fact that some patients who are frightened and totally unprepared have short and uncomplicated deliveries, while placid and apparently calm individuals can, and not infrequently do, have any of the forms of incoordinate uterine action. The exact mechanism whereby emotional factors influence uterine action is not known, but the most likely is by sympathetic nervous impulses, or by the action of adrenaline liberated by the adrenal medulla. Although there may be some degree of psychological control of uterine action, it does not seem to be the only operative factor.

Early rupture of the membranes, intra-uterine manipulations, and oxytocic drugs have been cited as factors causing this condition. It is possible that they are predisposing factors, but labour is frequently induced by just these means without causing ring formation.

Cephalopelvic disproportion is neither an active nor a predisposing factor, but the two conditions may exist simultaneously.

In summary, it would seem at this stage of our knowledge that the constriction ring is a primary functional disturbance of the uterus. It is probable that more than one factor may be operative, such as some form of physical interference in a psychologically unstable person, as suggested by Louw.[6] It is possible that the sites of constriction are based on an atavistic inheritance from the uterus of rodentia, carnivora and ungulata. The physiology of smooth muscle and of the factors controlling labour hold the key to the solution of this problem.

DIAGNOSIS

It is rare for the diagnosis to be made in the first stage of labour;[10] as a rule it is finally made only on vaginal or intra-uterine examination. There are, however, a number of pointers which, if taken into account as soon as they arise, will lead to the suspicion that all is not well. The appropriate examination will disclose the diagnosis, and thus the correct treatment can be instituted at the earliest possible time.

Symptoms

1. The manifestation by the patient of a decided change in her response to uterine contractions during the course of a normal, but prolonged labour. She complains of severe pain over the upper part of the uterus and senses that her pains are not effective.

2. Multigravidae may observe that the contractions differ from those of previous labours and that the pain starts before the onset of the contraction and persists after its cessation.

3. Backache, rectal pain, and pressure are often prominent when the constriction ring is low down.

Signs

1. A depression at the site of the ring may be seen and felt, particularly in a thin patient.

2. The upper segment is said to be circular and the lower cylindrical.

3. Often the diagnosis is made when forceps are applied and the baby cannot be delivered even with strong traction. The head will then be seen to recede quickly after each traction with the forceps. Sometimes the ring will be found when some intra-uterine manipulation is attempted, e.g. an internal version.

4. A definite diagnosis is made only on vaginal examination or even on intra-uterine examination. The following features are found:

(*a*) There is no descent of the presenting part during uterine contractions; in fact the presenting part often appears to recede with each contraction.

(*b*) The presenting part is loose in the pelvic cavity during and between contractions.[11]

(*c*) There is little or no caput succidaneum formation or moulding of the foetal skull.

(*d*) The cervix is thick and hangs loose, 'like a curtain' in the vaginal vault. It is not well applied to the presenting part.

(*e*) Owing to laxity of the cervix, a hand can often be passed upwards into the uterine cavity to palpate the ring.

Constant awareness that the condition does occur, is the single most important factor in early diagnosis, as in all other complications of pregnancy and labour that are not common.

The following case summaries illustrate the above features.

Case 1

Mrs. K. O'H, a 20-year-old primigravida, was admitted in labour, having had no antenatal care. She had been in the second stage of labour for 2 hours, with no advance of the presenting vertex. The cervix was found to be fully dilated and the vertex was 1 cm. below the ischial spines, but appeared to recede with each contraction. There was no moulding, but a small amount of caput formation was present. Under a general anaesthetic a constriction ring was found around the infant's neck, Neville Barnes forceps were applied, and inhalations of amyl nitrite were given. With minimal traction, a live male infant, weighing 7 lb. 9 oz., was delivered.

Case 2

Mrs. S. F., an 18-year-old, booked primigravida, was admitted in labour at term. The membranes had ruptured at the onset of labour, and she continued in an ineffectual type of labour for 72 hours, during which time the cervix dilated to 2 fingers. A deep depression now became visible and palpable above the symphysis pubis. In spite of conservative measures, the ring persisted, and at Caesarean section a live infant weighing 8 lb. 1 oz. was delivered. The ring was still present at operation.

Case 3

Mrs. L. M., a 29-year-old gravida 5, para 4, was admitted in labour at term. All her previous labours had been normal and she had been booked for delivery in the district. After 12 hours in labour the cervix was 4 fingers' dilated, but a deep depression was noted at the level of the umbilicus. The vertex receded with contractions and the cervix was thick and poorly applied to the head. Conservative measures had no effect on the ring and at Caesarean section a live female infant weighing 9 lb. was delivered. A classical Caesarean section was necessary in order to sever the ring and free the baby.

Case 4

Mrs. J. D., a 36-year-old gravida 10, para 8, was admitted at term as a non-booked case after having been in labour for 13 hours. Examination revealed a shoulder presentation, with the membranes

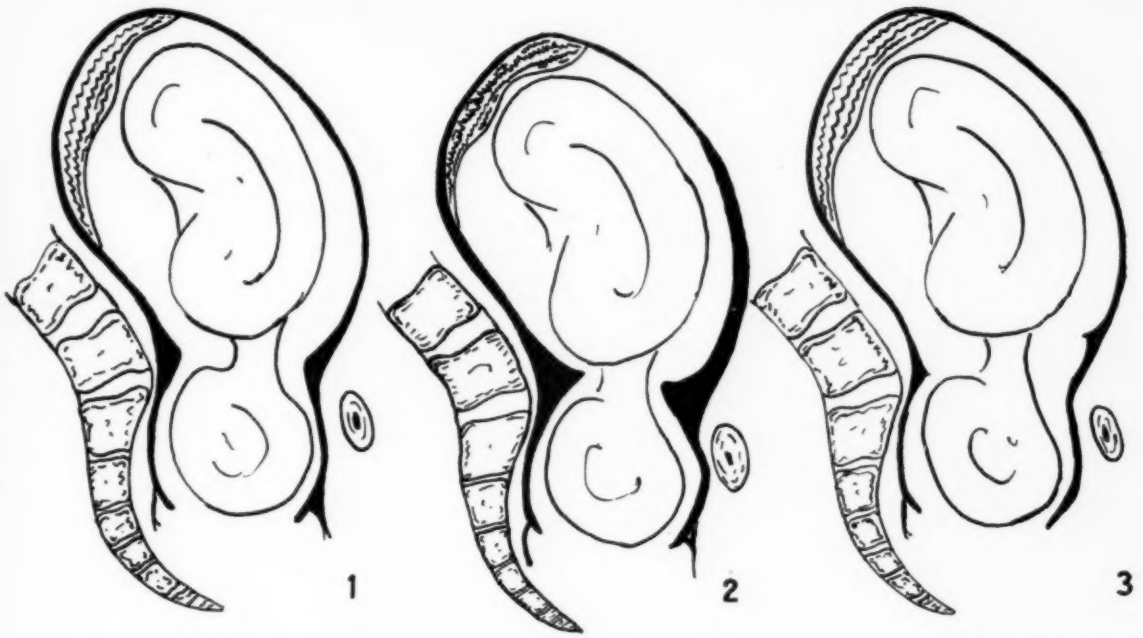

Fig. 1. Reversible constriction ring. *Fig. 2.* Irreversible constriction ring. *Fig. 3.* Bandl's ring.

intact and the cervix almost fully dilated. Under a general anaesthetic an internal version was performed and, since the cervix was now fully dilated, a breech extraction was commenced. Difficulty was experienced after bringing down a leg, and on internal palpation a tight constriction ring was felt below the breech and around the leg which had been brought down. In spite of amyl nitrite and deep chloroform anaesthesia, the ring remained and was still present at Caesarean section when a live male infant weighing 7 lb. 3 oz. was delivered.

Case 5

Mrs. K. D., a 33-year-old gravida 3, para 1, was admitted as a booked case at 36 weeks for the treatment of pre-eclampsia. She did not respond to the treatment prescribed, and it was decided after 1 week to terminate the pregnancy. Labour was induced by a combination of medical and surgical inductions, followed 12 hours later by a pitocin drip. After 28 hours of poor labour, the cervix was fully dilated, but there was no advance of the presenting vertex, which in fact appeared to recede with each contraction. Under a general anaesthetic a constriction ring was found around the infant's waist, amyl nitrite inhalations were given, and an easy forceps delivery of a 5 lb. 5 oz. female infant was performed.

PROGNOSIS

In 1935 Rudolph[4] collected 371 reported cases from the literature. The maternal mortality was 15% and the foetal mortality 46%. In no less than 97% was some form of operative interference necessary. The reported maternal mortality varies between 10 and 50%,[4,12-14] and the reported foetal mortality between 40 and 80%.[14] In a series of 56 cases reported by Louw[6] the foetal mortality was 30%, and Rudolph and Fields[15] recorded a maternal mortality of 3·5%, while Rucker[16] lost only 1 mother in 270 so affected.

From these figures it would appear that, although the maternal mortality has diminished in the past 20 years, there has not been the expected improvement in the outlook for the infant that should have been associated with the rising standard of antenatal and intranatal care.

TREATMENT

A. *When Diagnosed in the First Stage of Labour*

Intelligent expectancy should guide the attendant here, and all efforts should be directed against allowing the mother to become exhausted and despondent:

(*a*) Adequate sedation in the form of morphine, gr. ¼, or pethidine, 100 mg., should be given.

(*b*) Fluids by intravenous infusion must be given where indicated, and therefore a strict watch must be kept for commencing dehydration and acidosis.

(*c*) An antibiotic cover should be started early as a prophylactic measure.

The condition is rarely diagnosed in the first stage of labour as emphasized by White.[12]

B. *When Diagnosed in the Second Stage of Labour*

Greenhill[17] states: 'When constriction ring dystocia persists during the second stage of labour, this stage may last for 24 hours or more, and the uterus will not rupture spontaneously, regardless of the duration of labour, if the cephalopelvic relation and the position and presentation are normal'. This is a point of view on which many would not agree, holding that once the cervix is fully dilated steps should be taken to effect immediate delivery of the infant. There is no doubt that the longer the second stage is allowed to continue, the higher will be the foetal mortality and morbidity.

Conservative measures should always be tried initially. These include:

1. Amyl nitrite inhalations, which will have the desired effect on some occasions.

2. Adrenaline, 8 minims, by slow subcutaneous injection may be of value. However, Kaiser and Harris[18] emphasize that this substance in high concentrations is oxytocic through-

out the uterus and has very marked systemic effects. In low concentrations it is said to be inhibitory to uterine activity and probably has no systemic effects. It has the added advantage of not being a respiratory depressant for the child.

3. Deep general anaesthesia, preferably with chloroform or ether, which are sympathomimetic drugs, should be tried and may have the desired effect. Cyclopropane, a parasympathomimetic drug, should never be used.

4. Magnesium sulphate, 2 ml. of a 50% solution, or magnesium gluconate, 10 ml. of a 20% solution, are recommended by Abarbanel.[19] The magnesium is said to act directly on the myometrium in slowing the rate of the contraction wave. Calcium chloride should always be on hand in case of collapse of the patient.

5. Caudal anaesthesia has been used, but its effects are completely unpredictable and variable. The muscle relaxants have no effect whatever on a constriction ring.

Generally speaking the constriction rings which will respond to conservative measures are of the reversible type, and will do so within a relatively short period of time. These measures should not be continued with indefinitely in the hope of avoiding a Caesarean section, for prolonged efforts invariably endanger the life of the infant. It is far better to deliver a live baby by Caesarean section, than a dead or dying baby by the vaginal route.

Should the conservative measures be successful and result in relaxation of the ring, spontaneous delivery, forceps delivery, or breech extraction can easily be performed—depending on the circumstances pertaining at the time.

Vaginal delivery should not be undertaken until the ring has relaxed. Forceps delivery, version with or without extraction, craniotomy, embryotomy, and Dührssens incisions are not only contra-indicated, but are extremely hazardous.

The Rôle of Caesarean Section in this Condition

If the ring cannot be overcome by an adequate trial of the above measures, and with the infant still alive, Caesarean section should immediately be performed. The present-day safety of the operation, with antibiotics, blood transfusion, and improved anaesthetic techniques, makes it the procedure of choice under these circumstances and where there is any added complication, e.g. pre-eclampsia, questionable disproportion, or infertility in an elderly primigravida.

With a dead infant, heavy sedation, general resuscitative measures, an antibiotic cover, and patience will frequently result in relaxation of the ring and vaginal delivery. Consideration of the foetus does not arise here, and time is on the side of the attendant. It is as well to remember that Caesarean section may even be necessary under these circumstances in the interests of the mother, and that this condition is one of the rare remaining indications for a classical Caesarean section, where the foetus is tightly gripped by a constriction ring in the upper segment.

Constriction Rings in the Third Stage of Labour

The separation of the placenta and its delivery are dependent on contraction and retraction of the upper uterine segment. As the trunk of the foetus leaves the uterus, retraction occurs, and it is believed that the placenta is separated from its bed within 1-5 minutes.[20] It does, however, take 10-15 minutes before it descends to the lower uterine segment, possibly due to adherence of the membranes. The signs formerly associated with 'placental separation' should perhaps be more rightly termed 'signs of placental descent'.

Any upset in the normal action of the uterus will lead to incomplete separation of the placenta, or retention of the separated placenta with the attendant dangers, chiefly postpartum haemorrhage. Although one cannot recognize the various individual forms of inefficient uterine action at this stage, constriction ring falls into this category, and an 'hourglass contraction', as it is often called, will interfere with the expulsion of the placenta.

Probably the main cause of this group of conditions is interference with the fundus of the uterus and premature attempts to express the placenta. The early administration of oxytocics has been incriminated, but this contention has never been proved.

The treatment of a constriction ring at this stage does not as a rule present any difficulty. Manual removal of the placenta, with manual dilatation of the ring where necessary is always successful, although amyl nitrite and deep anaesthesia may be necessary at times.

SUMMARY

1. The types of constriction ring encountered in labour are described and defined.
2. Their aetiology is briefly discussed.
3. Their symptoms, signs, prognosis, and treatment are reviewed.
4. A few illustrative case histories are briefly described.
5. A plea is made for early diagnosis and prompt treatment.

The treatment should be active and radical where necessary, and it is contended that only in this way will the present very high mortality rates, both maternal and foetal, be reduced.

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PHILOSOPHY IN A PETRI DISH*

DAVID ORDMAN

Does it seem strange to consider philosophical questions in a journal born in the laboratory? It may be that the mere dry bones of laboratory work are—mere dry bones; but no laboratory activity has any meaning unless thought enters to impart that spark which is life. In the world of bacteriology what do we cultivate on a Petri dish? If bacteria only—then the true laboratory

* Reprinted from Newsletter of the SAIMR, April 1961, vol. 8, No. 1. Published by the South African Institute for Medical Research, Johannesburg.

function is dimmed. If you cultivate philosophy on a Petri dish you surely see more than the colonies; a living growth of positive thought reveals itself to stimulate and refresh. For after all, are not bacteria living things—and of a social kind! Who has heard of a single lonely germ appearing on the plate!

And so it is not only with the Petri dish, but with that vast interesting phenomenon we call the laboratory. Indeed, is not the whole of life a laboratory where consciously or unconsciously we become entangled in nature's experiments? Does not contact with other human beings or situations constitute a series of actions and reactions? Can we be confident in advance of responses or emotions which a particular set of circumstances will invoke in us?

Laboratory life is a life of adventure—explorations, huntings, discoveries, excitements. You trap bacteria, harmful and otherwise, on an agar plate. Your trapping may not be successful, so you set a new trap—a medium of a different kind. You cannot work with tools and apparatus blindly. It may be subconsciously, but you are constantly thinking not only of a particular germ or chemical or tissue or force, but also of the relation of these to yourself and to your fellowman. Is it enough to say that there is albumin in the urine, or do you wish to know why the albumin is there and how it got there? When the report of 'albumin present' is issued, the task is not completed—it is only just beginning, not for you perhaps, but, at least, for a fellow human being.

There is a certain fascination in the medical laboratory: the fascination of another world, the microcosm of living bacteria—their unimaginable numbers and their unbelievable powers. To turn the objective onto a field is to enter a wonderland that Alice did not dream of. It is easy to become blasé, to take these worlds in our stride, but in so acting we lose that child-like freshness of outlook that keeps man young. Parasites are a nuisance to you when they make you ill—but you are a nuisance to parasites when you die; your death imperils their existence. It is give and take in the world of nature and, looked at objectively, is it not a little difficult to determine the relative importance of the *Bacillus coli* and you!

It is a privilege to take part in laboratory life. It is more than a job; it is a relaxation from the artificialities of an outer world. It brings you into contact with nature, it provides you with the opportunities of delving into her secrets. The outlook is limitless—the horizon is never-ending. Soul-hunger can be satisfied at the laboratory bench; for in the search for truth you find beauty, and truth and beauty are happiness. You manipulate worlds with a platinum loop; you control the destinies of animals in your biological experiments; you are the wielder of an immense power, and, are you worthy of it? You help in the advancement of knowledge and play your part in the interrelationship of the creature and its host. You collect the facts for speculation, you assist in the solution of the problems—philosophical and material—of the welfare of mankind.

The Patient First!

In the drama of human health the principal actor is our fellowman—the patient. As the efforts of the doctor are directed to the well-being of his patient, so, too, from the laboratory point of view, the patient comes first. As the doctor must needs give of his best to the patient, so the laboratory worker is solemnly bound to give of his best. In a medical laboratory, however routine the work may appear to be, each specimen represents an individual human being with hopes and fears and pains and passions like yourself. Specimens are sometimes received in the laboratory, taken from the patient, which are obviously unsatisfactory in type or variety or manner of collection—unsatisfactory, that is to say, in providing the best for the patient. Is it our business to carry on and get the job done, or is it wiser to direct attention to what we as laboratory people consider deficiencies. Is there not sometimes a mental struggle suspecting a possible resentment at our interference! Experience, however, reveals that the physician is almost invariably appreciative of advice from the laboratory, for laboratory services are specialized services, and the medical practitioner is seldom also a laboratory specialist. In any event, with the laboratory slogan of 'the patient first', the signpost to where duty lies is not far to seek.

Is the Laboratory a Machine?

The danger of the laboratory becoming an automatic, fact-finding mechanism is always present. But you may say, as we pay the barber for a shave, the tailor for a suit, so the patient pays the laboratory for the result of an investigation, and that is all there is to it. In a sense that is true, but the laboratory worker is in the peculiar position of noting, not only the results of an investigation for one doctor, but also of thousands of investigations for hundreds of doctors. In other words, you score over the individual physician in that you have bulk of material to deal with and so are in a position to compare and contrast. There is always the possibility of finding a better or more economical way of arriving at the same result or indeed at a more accurate or more worth-while result. Has any laboratory process reached perfection, no matter how simple the procedure?

In one sense though, a medical laboratory is indeed a machine in which we, as individuals, are the cogs. And the cogs must fit and integrate harmoniously, for the machine will not function if a cog breaks down. If one department fails in some particular respect, Mr. Jones will not be properly treated for a bacillary infection of the bowel or Miss Smith for a blood condition. There are no water-tight compartments in the medical laboratory, and cooperation and collaboration are of necessity the watchword. The doctor is interested in the final picture presented by the various laboratory reports he receives, and probably does not appreciate the preliminary basic work and experience that have gone to make a laboratory diagnosis a matter of a few hours or even a few minutes. But this basic work, this fundamental background, is not made of loose rubble, but of rock cemented together by departmental cooperation and held in place by intelligence. Cooperation and collaboration result in a far greater thing than the mere issue of a report; there is the meeting of minds, the exchange of experiences and the healthy friction that emerges therefrom. Friction of this kind is a good thing—it lets truth appear. No one has the monopoly of the right point of view; indeed, who is to judge what the right point of view really is! But the more comprehensive the outlook, the more receptivity there is of the other man's viewpoint, the greater the possibility of attaining that completeness which approaches truth.

Accuracy

Even if the chemical laboratory is rapidly approaching it, the biological laboratory has not yet reached the factory stage where results are obtained by machine. Whether a thousand tests have to be done or one test, some person has to do them and interpret the findings. You cannot yet pass a series of tubes of blood or pieces of tissue through an apparatus which will automatically record the more subtle biological findings or pathological conditions. It is vital in bulk work to avoid blunting accuracy. The results of multiple tests must equal in accuracy that of a single well-conducted test. But bulk work introduces monotony and fatigue, so precautions are necessary to counter these bugbears. Accuracy dare not suffer on any consideration; if it does the whole function of the laboratory is lost. The factors tending to reduction of accuracy in laboratory work must at all costs be rigorously searched for and eliminated.

Teamwork and the Individual

What is the importance of teamwork in laboratory life? In some organizations and in the conduct of certain experiments, a set of specialists is put on to an investigation so that a particular subject is investigated fully from all angles. There is little doubt that, by this method of the properly coordinated expenditure of energy and labour, a maximum of results is derived with greatest human economy. It is anything but certain, however, that this is the method that will produce great thinking—great in the sense of brilliant or epoch-making, a 'break-through' of current conventional concepts. A committee can certainly decide upon a procedure, formulate its findings and evolve a mode of action. But can you conceive of the combined brains of persons sitting in solemn conclave giving birth to a profound idea! Can a committee write a poem, compose a sonata, or paint a picture? After all, the great ideas in the history of mankind were born in the minds of individual men and not as a result of teamwork. Consider Moses, Jesus, Mohammed, Newton, Darwin, Pasteur, Einstein and others who have, as it were, lifted mankind from one developmental plane to a higher level, not by a gradual evolutionary process, but in a jump. It is indeed given to few of us to provide a new and vivid slant to the outlook of the human mind or to flash a new vision before mankind. But many of us have the privilege, in a smaller way, in more humble circumstances and

to a relatively small community or even to one other individual, of giving a new point of view, of formulating an idea, of crystallizing and giving substance to vague conjectures. The artist in his painting teaches us to see things we have perhaps not noticed before; he directs our eyes upon an aspect of life and matter previously overlooked.

How Sacred is Authority?

A 'point of view' is not an ephemeral matter, but the resultant of the conflicting material and spiritual forces of a lifetime. A point of view is based on training and experience, and the complex, multiple influences that mould man to what he is. It is obvious, therefore, that an honest point of view deeply held is a precious thing, a treasured possession, and must contribute to the progress of the society in which we live. We are in peril when our point of view wavers—not when it wavers in the presence of convincing and reasoned demonstration, but when it is weakly aborted in the presence of an authority. So many permit their point of view to emerge as a still-birth or a weakling because an authority has held otherwise. In this sense authority and the all too often cramping influence of tradition act as brakes on progress, and scorch with their baleful gleams the tender shoots of thought and action in that great mass of population, half contemptuously referred to as the 'average man'. The average man is a myth; there are no average men. Each man is an individual—an entity in whom life's forces flow. A million machine-made harps struck in the same way by the same force will produce an identical note. There are no identical notes in that fluid, plastic, mobile collection of ever-changing personalities we call man. But the herd is there all the same—that mass of human beings, struggling, eating, loving, living, blown about in particular directions by the wind of authority, by the forces of convention, by the pressure of tradition. He is a strong man who fights these forces and preserves his individuality. But it is these strong men who are the doers, the creators, the salt of the earth! They are the worth-while. They are the instigators of progress. They constitute the axis on which the world revolves. It is unjust to personality to permit oneself to be swamped by the heavy-handed dictum of authority. Let your point of view prevail until it is untenable. No man should be so enmeshed in the coils of the complex of inferiority that he does not raise his voice because it is written otherwise in a book, or said otherwise now or in the past by another person looked upon as an authority. Why, the literature of science is littered with the discarded theories of authority! In physics, most strikingly, concepts of matter change and change again and have not yet reached finality. Medical books are monuments to the outworn thoughts of tradition. Laboratory people, no less than others, should not allow themselves to be dragged along at the wheels of the chariot of authority, but should leap into the driving seat and think for themselves!

This surely does not mean ignoring or overlooking the work of others, but it does mean positively and critically and even sceptically evaluating the productions of contemporary and past authorities—and building on them if found good. In research work it can be a matter of opinion whether it is wise first to soak oneself in the findings of other people on the same line of investigation and to commence from there. Some consider it better to approach a problem with a fresh eye and an unbiased outlook, and to settle down to read the work already done on the same subject only after a thorough consideration of the whole matter from one's own view-point. There is a danger, however, in over-reading, for it tends to throw the mind into a groove and dim the vision.

This desire not to accept authority at its face value is a good thing for another reason. It promotes experimental work in repetition of that of others and, in stimulating the critical faculties, helps to elaborate the truth. To confirm a finding is a satisfaction, to differ from it a stimulus, but to go in pursuit, to determine the 'how' and establish the 'why' of these differences brings a joy to the heart of the experimenter. It is in divergent results that clues lie—clues perhaps to problems even greater than the ones under investigation. And, more especially in the laboratory, it is tragic to permit the unusual to be neglected. For the unusual finding may be that by-path waiting to be explored. It is only too common for the unusual to be explained away vaguely as an error in technique, perhaps, or even worse, shrugged off with a deprecatory gesture at one's fallibility. That inferiority complex rears its sombre head again and should be ruthlessly fought.

Experimentation and the Statistician

It is interesting to consider the methods of experimentation in the medical laboratory world. The 'bulk' method involves large numbers of trials, and it is tempting to conclude that the finding is the correct one if it is supported by the comforting phrase 'statistically significant'. It is perhaps subtler to design an experiment which shall be crucial. Thus, a thousand guinea-pigs may be sacrificed to determine the prophylactic or therapeutic value of a substance, but a crucial test or a series of such tests may be arranged to give the required information with far less animals. And yet biological work in the laboratory entails a profound appreciation of the fact that animals, unlike chemicals, have their individual idiosyncracies, and that conclusions from inadequate numbers of tests are apt to mislead. The transfer of results of animal experiments to human application may be fraught with danger. And, turning the situation around, it is sometimes only too difficult to find a suitable animal in which the human disease will be represented more or less in the human way, if at all!

There is a humbling and salutary lesson for the laboratory worker to be found in the trenchant criticisms of the statistician. It is desperately easy to slide smugly down the path of data-accumulation and result-finding, either in diagnosis, prophylaxis, or treatment, but the correct evaluation of the findings demands a respect bordering on reverence. And to those without the mathematical flair, let your conclusions be guarded until the statistician has stopped frowning. They say that anything may be proved by statistics, but it is more true and less flippant to assert that too many things can be demonstrated without statistical control.

Beware of Finding what you Seek!

When the mind is ardently focussed on an objective, cool judgment comes tardily and enthusiasm may bring into existence that which does not exist. Have you searched intensely for virus bodies and found them with a whoop of joy to be quenched by the chilling glances of a colleague, not so interested, who sees just debris? Have you inoculated white mice with material from 'pink disease' and found pink feet in these animals, only to discover when your excitement has waned that the feet of white mice are pink anyhow? How easy it is to be swayed by your desire, how difficult to preserve a judicial calm undismayed by a fruitless quest. But negative results are as valuable as positive results—they smooth the way for the searchers that follow. They are a contribution to knowledge, and he is wise who records them with honesty and objectivity. You may find what you look for, so beware! Curb your thrill with a sigh, and look again!

Depression in the Petri Dish

But there is another humbling thought in the bacteriological laboratory in particular, and that is: when you have planted your specimen on the standard media recommended for that purpose, what assurance have you really that the organisms found are genuinely all that are present in the specimen or causally associated with the morbid condition under investigation? Think of the many germs that grow differently on different media and that grow on some and not on others. Think of the influence on germ growth of variations of pH value. Consider the influence on germ cultivation of a carbon-dioxide atmosphere, of a moist atmosphere, or of no atmosphere at all. How little is known of the germs that might be revealed by anaerobic cultivation of specimens, and of the significance of organisms that do come up in these circumstances. What is the real truth about the bacterial flora in secretions and excretions in different pathological conditions? How often have you searched for a possible virus infection in specimens sent to you? You report with confidence that the *Staphylococcus aureus* is present in a sample of pus. It no doubt is so, but surely the report should read:

> 'The *Staphylococcus aureus* was recovered from this specimen on agar medium of a particular concentration, made with a particular brand of peptone and a certain pH value, after growing for a period of 24 hours in an incubator designed to keep the temperature constant at 37°C. We do not know what organisms would appear if we grew the specimen in semi-solid medium or *in vacuo* or in carbon dioxide or in the yolk sac of an egg or in the peritoneal fluid of a guinea-pig, the brain of a mouse or the muscle of a fish, or under anaerobic conditions or in a medium which we do not yet know, but which we may find out one day to be most suitable

for the growth of an organism which may be in the specimen, but which we have not recovered because we do not know quite how to look for it.'

Of course this is exaggeration, but the trend of thought will be obvious. And yet there is so much exaggeration! The physician is as ignorant, or else he would chuckle and say: 'your fantastic report is right; a vaccine was duly prepared from the *Staphylococcus aureus* on the basis of your report and the patient still enjoys the company of his boils'.

We are proud of our laboratory and realize what a clever handmaiden to medicine it is, and yet how crude our knowledge still is with regard to bacterial physiology and the interrelationship of parasite and host. It is sometimes depressing to philosophize over the Petri dish and wonder: are they all there or have some important elements escaped our vigilance because our handling is so relatively primitive? What do we see with our high power lenses? Already a start has been made, but we are still at the beginning of seeing the internal structure of bacteria. Perhaps we shall laugh one day at the crudities of our present microscopic examinations. In the associated science of immunology we are only now at the threshold of the knowledge of immunizing processes and the chemistry involved. Does it not seem crude to inject the bacterial bodies in vaccine form instead of the refined essential chemical products extracted from these bodies? Does it sound so advanced to you to inoculate a child therapeutically with the blood serum of an immunized horse, rather than with essential clearly-defined specific chemicals constituting the antibodies!

Hope in the Petri Dish

It is fascinating to be in a laboratory if only to see in it a mirror reflecting the general trends and tendencies in modern medicine. A new fashion in diagnosis or treatment and lo! the laboratory is flooded with specimens for specialized study. The sea of medicine billows and swells with new currents in medical thought—drugs, endocrines, vitamins, and each at its crest carries with it, in the breasts of the lay and medical public, the hopes for a disease panacea at long last. But the wave crashes and a new wave comes and the sea remains sea and the navigators thereon remain—doctors. The song of the antibodies echoes around the world. Infectious diseases are being beaten, nutritional diseases are nearly conquered. Assuredly with further developments bacterial and parasitic monsters of disease will find their fate, and metabolic diseases will slink out, vanquished. It is a grand thought, but perhaps it is a little difficult to visualize the Utopia of 'No complaints'. Is it possibly a little depressing to wonder if the goal of human happiness will be attained when mankind may idle with impunity and overindulge without retribution. Will there be drugs to stimulate the production of self-control, vitamins to tone the system against selfishness, and hormones to regulate the flow of the milk of human kindness!

But in the great laboratory of human life all is possible, and the experimenters in health and happiness must always come back to work and philosophize over the Petri dish of mankind.

A HOSPITAL ADMINISTRATOR THINKS ALOUD*

P. N. SWANEPOEL, *President, Northern Transvaal Branch (M.A.S.A.)*, 1960

My address to you tonight will not be on diseases or new advances and techniques in medicine, or any scientific subject at all, but mainly on ourselves. When I say ourselves, I refer to the relationship of the doctor, whether he is a specialist or a general practitioner, to the hospital where he treats his patients or to which he sends them for treatment.

Dr. Swanepoel

Let me say at the outset that, although I am about to express critical remarks about the profession, I am as much part of that profession as you are. If any one person is aware of the deteriorating patient-doctor relationship, that person is the medical superintendent of a hospital. He is the person who receives all the complaints against the doctor, and the hospital, and has to find the answers. In the search for these answers, he obtains an intimate knowledge of his profession and how the profession functions.

Unfortunately it is not only he who obtains this knowledge, but also the lay persons on the staff of the hospital. We are apt to forget that the administrative staff of the hospital is composed for 98% of lay people, and it is not possible to prevent them from having access to the medical records and correspondence of the hospital. Included among the lay staff of the hospital are the nursing assistants and nursing aids. These two groups come into even closer contact with the profession by nature of their work in the wards. Some of them will sometimes freely discuss what they hear and see in hospital, although they are warned at the commencement of their duties that talking out of hospital will result in instant dismissal. Do we as a profession realize that a doctor's ability as a surgeon or his proficiency as a diagnostician is often made in the ward where he treats his patients daily? Members of the nursing staff are often consulted outside the hospital by would-be patients about the capability of Dr. X as compared to Dr. Y. What is said respectively of Dr. X and Dr. Y by the nurse, or for that matter by any lay person on the staff of the hospital, will depend on his relationship with members of the hospital staff, his skill in the theatre, and his approach to his patients in the ward. Here in the hospital you are always on test. You cannot afford to 'go easy'.

We, as a profession, must realize that while in the hospital we are always in the centre of the spotlight, and the shadow cast by us on the wall will exaggerate all our shortcomings and mannerisms for all to see.

I shall now discuss some of the incidents that are fairly common in the relationship of doctors to members of the hospital staff. These incidents often place either the doctor or the hospital in a most embarrassing position.

Voorvalle

Daar is byvoorbeeld die geval waar 'n welbekende spesialis die vrou van 'n vooraanstaande persoon in die politieke wêreld gehad het as pasiënt. Volgens die spesialis was die verpleging van die pasiënt nie van die beste nie. Wat doen die spesialis toe? Hy kom nie die matrone of die superintendent oor die aangeleentheid spreek nie ten spyte van die feit dat hy bekend is met die regte prosedure aangesien hy al jare lank in die personeel van die hospitaal dien en die prosedure al voorheen gevolg het. Nee, hy sê vir die eggenoot van die vrou dat daar niks is wat hy verder aan die saak kan doen nie, aangesien hy al met die betrokke suster gepraat het. Hy gee nou die saak gewonne. Hy, die eggenoot, moet nou maar kyk of hy iets kan doen. Die eggenoot het my gebel en padlangs beduie wat hy dink van die hospitaal. Is dit samewerking? Mag ek u nou net vra wat die betrokke spesialis daarvan sal dink as hy miskien eendag nie sy pasiënt besoek nie, of vergeet om iets te doen, en die suster sê aan sy pasiënt dat sy nie weet waarom pasiënte na dié dokter toe gaan nie omdat hy altyd vergeet om hulle behandeling op te skryf of om sy pasiënte te besoek? Goeie verhoudings tussen dokter en hospitaal kan net behou word as dit van altwee kante kom. Ek kan u verseker dat die interpretasie wat aan die voorval gegee is, 'n swak refleksie op die spesialis werp, naamlik, dat hy probeer het om 'n indruk te maak. Ek kan u verder verseker dat die spesialis miskien 'n indruk op die pasiënt gemaak het, maar sy status as geneesheer het sekerlik verminder in die hospitaal in daardie afdeling.

In die onderhoud met die eggenoot, is die hospitaal-owerheid in die moeilike posisie geplaas om die saak tog regverdig te stel, en om ook nog die dokter te beskerm deur nie te laat deurskemer

* Valedictory address, Pretoria, 9 February 1961.

dat hy wel goed bekend was met die regte prosedure nie en dat sy optrede, selfs al was daar 'n gegronde klagte, verkeerd was. Ek wonder of hy besef dat, as ons aan die eggenoot vertel het wat regtig gebeur het, hy nie miskien sou gedink het dat die spesialis versuim het om sy plig te doen nie?

Kom ons neem nou die geval van 'n private pasiënt wat in dieselfde saal verpleeg word as 'n hospitaal-pasiënt. Dit gebeur soms dat die private geneesheer nie sy pasiënt op 'n sekere dag besoek nie, terwyl die ‚vry' pasiënt 'n paar besoeke op daardie dag ontvang, naamlik, deur die hoof op 'n saal-rondte, die kliniese assistent, en die huisdokter. Die private geneesheer vergeet miskien die volgende dag om aan die pasiënt te verduidelik waarom hy nie gekom het nie. Ek kan u verseker dat die volgende moeilike vraag dikwels aan die suster gestel word: ‚waarom kry die „vry" pasiënt meer besoeke as ek?' Wat moet ons wat in die hospitaal is, antwoord?

Dan is daar ook die geneesheer wat in sy geselsie met die pasiënt 'n nuwe behandeling voorstel as 'n moontlikheid, maar wat nooit die opdrag aan die suster gee om dit uit te voer nie en die voorskrif ook nie opskryf op die bedkaart nie. Môre-oormôre vra die pasiënt daarna en dan word met verbasing gevra: ‚Suster, het hy dan nog nie die behandeling gegee nie?' Wie moet die pasiënt dink is nou die sondebok? Laat ons eerlik wees oor die hospitaal teenoor ons pasiënt. Moenie beloftes maak wat teenstrydig is met die reëls van die hospitaal nie. Besoekure is spesifieke tye en geld vir alle pasiënte, of hulle nou ook al hospitaal-pasiënte of private pasiënte is. Moenie vir die moeder sê sy kan klein boetie enige tyd kom besoek, net om haar te troos en om boetie in die hospitaal te kry nie. As die hospitaal sy reëls toepas dan word dit aan ons gesê: Dokter het gesê ek kan dit en dat doen in die hospitaal. Die dit en dat mag teenstrydig wees met die reëls, en elke dokter moet bewus wees van die reëls. Goeie verpleging kan alleen geskied as elke dokter die hospitaal help om die beste verpleging aan die pasiënt te gee deur in alle opsigte saam te werk met die hospitaal.

Ons weet dat ons hier is om die diens te lewer aan die publiek. As u 'n dringende geval het wat toegelaat moet word, moet u daarop bedag wees om nie aan die naasbestaandes van die pasiënt of aan die pasiënt beloftes te maak wat moontlik nie uitvoerbaar mag wees nie. Dit lei net tot onaangenaamhede. Die hospitaal word telkens gedreig met 'n hofsaak omdat 'n pasiënt nie die behandeling ontvang het wat die dokter voorgeskryf het toe hy die pasiënt na die hospitaal toe gestuur het nie. Dit gebeur dikwels as pasiënte tot sterwe kom. Die volgende voorbeeld is tekenend in hierdie verband: 'n kind met 'n bloedsiekte is as 'n noodgeval toegelaat. Die belofte van die dokter aan die moeder was dat die kind onmiddellik 'n bloedoortapping sou kry by toelating. Dit was 'n hospitaal-geval. Die kind is 'n uur na toelating oorlede. Die hospitaal is van nalatige behandeling beskuldig omdat die bloedoortapping nie onmiddellik gegee is nie. Die ondersoeke wat egter gedoen is toe die geval ingekom het, het aan die lig gebring dat dit een van die bloedsiektes was waar 'n bloedoortapping van geen nut kon wees nie; dit kon die toestand eerder vererger het. Die beskuldiging van nalatigheid word gesteun deur die geneesheer se belofte. Wat staan ons te doen om ons saak te regverdig, sonder om die reputasie van die geneesheer te beskadig? Die hospitaal het al die fasiliteite om die regte diagnose vas te stel en die daaropvolgende behandeling so spoedig moontlik toe te pas. Moenie sake kompliseer deur in sulke gevalle voor te skryf wat om te doen nie, want in u haas mag u moontlik nie in staat wees om al die nodige toetse te doen nie, en u diagnose kan ook verkeerd wees.

Ek wil nie beweer dat die hospitaal nie ook soms skuldig is nie. Sekerlik maak ons foute, maar graag wil ek weer vra dat ons die foute aan die matrone of superintendent moet rapporteer. As dit 'n kollega is, gesels self met hom. Dit gebeur wel dat ons ongevallebeampte soms 'n verkeerde diagnose maak of 'n faktuur nie raaksien nie. U sal miskien in die nag of oor die naweek uitgeroep word na die pasiënt. Moenie die hospitaal voor die pasiënt kritiseer of u kollegas se vaardigheid in twyfel trek nie. Dink voor u praat. Het u nie ook al 'n fout gemaak nie? Ek verwag nie dat u die feite moet verbloem nie. Kom gesels gerus met ons, die owerheid, oor die geval, of nog beter met u kollega. Ons en hy sal dankbaar wees vir u advies, en dit sal help om te verhoed dat dieselfde fout weer begaan word. 'n Goeie gees tussen u, die hospitaal, en u kollegas is van die uiterste belang.

Die verpleging is nie altyd wat u wens nie. U moet egter ook onthou dat in 'n opleidingskool, verpleegsters opgelei moet word in die praktiese sy van verpleging, en daardie opleiding moet gedoen word op u pasiënte. Het u al daaroor nagedink dat die verpleegster, as sy begin, pas die skool verlaat het? Sy is miskien net 17 jaar oud. Die suster in beveel neem die bevele en is verantwoordelik daarvoor, maar dit gebeur soms dat die jong verpleegstertjie groot verantwoordelikhede op haar skouers moet neem na 'n jaar. Hier in die sale is sy altyd besig met pasiënte wie se lewens afhang van die behandeling wat sy aan hulle moet gee. U skryf net voor. Dink terug aan u studentedae en aan die eerste keer toe u 'n pasiënt moes ondersoek, of aan die eerste keer toe u as huisdokter 'n noodgeval ondersoek het en 'n beslissing moes gee. Selfs nog vandag as ervare praktisyn is dit soms moeilik. Hoeveel jaar was u nie ouer as die verpleegster en hoeveel meer ondervinding het u nie gehad voor u op u eie bene gestaan het nie?

Our profession has always been an honourable one. Let it stay that way. Let us not commercialize it and make it cheap. Patients who come to see us in our rooms and who need hospital treatment, should be carefully questioned to see if they can really afford the cost of hospitalization, including all the costs that go with hospitalization today. Explain to them, if you are in doubt about their financial position, that your bill will only be for part of the cost. If you feel that they cannot afford treatment as private patients, tell them so and tell them that they can obtain the necessary treatment as hospital patients. I feel that this is the correct way. Otherwise, they may be forced to come to the hospital to ask for a reduction of the hospital fees.

More examples could be enumerated, but I feel that enough has been said. Let us at all times strive to be worthy of our professional status and at all times try to make the hospital-doctor-patient relationship something that will be a credit to us all.

Ten slotte mag ek u net daarop wys dat dit nie my bedoeling is om die professie in 'n slegte lig te stel nie. Ek hoop en vertrou dat u my woorde slegs sal interpreteer as 'n waarskuwing dat dit tyd is dat ons as 'n professie moet nadink oor ons toekoms, en oor die daaglikse agteruitgang van die pasiënt-dokter verhouding. Wat gaan ons daaraan doen?

SUGGESTIONS FROM GRAVIDITY DATA OF CULICINES IN NORTHERN RHODESIA*

J. J. STEYN, PH.D., F.R.E.S., *Research Institute, Tzaneen, Transvaal*

INTRODUCTION

According to the Report of the Second Regional Conference on Malaria Eradication held at Addis Ababa in November 1959,[4] it is considered that the bio-assay testing of insecticidal deposits usually furnishes inconclusive results, and new biological methods of evaluation should be worked out. In order to obtain a better insight into their spraying campaign against malaria vectors, Dr. F. A. Donnolly, *O.B.E.*, M.B., Ch.B., D.P.H., Medical Officer of Health, Lusaka, and Mr. R. A. Mansfield, B.Sc. (Chem.), B.Sc. (Bact.), therefore decided to use the simplified gravidity technique for determining physiological resistance to BHC.[1] They have kindly supplied the following information for publication.

* Published with permission of The Secretary for Health, Pretoria.

BHC APPLICATION

An average of 2 lb. of 75% BHC, containing 10% of the gamma isomer diluted in 3 gallons of water, was used for spraying 1,000 square feet of hut walls and inner roof thatch. This is equivalent to about 75 mg. of active ingredient per square foot.

RESULTS

It is not possible to report at all regarding gravidity of *Anopheles gambiae* Giles, for the control situation is such that no single adult *gambiae* or *A. funestus* Giles has been recovered in the municipal area of Lusaka during the past 2 years.

Our assessment of BHC residual effectiveness has, therefore, of necessity to be based on 2 factors, namely, the absence of malaria

vectors within the municipal area (although prevalent immediately outside the boundary), and the degree of culicine gravidity.

During the past rainy season — the summer of 1959 - 60 — 394 adult culicine mosquitoes from 3 African townships, which were considered representative, were examined. Data were as follows:

Males	*Females*				*Total females*
	Unfed	*Fed*	*Half-gravid*	*Fully gravid*	
67	107	153	63	4	327

DISCUSSION

1. The total number of 394 culicines is considered too small to yield definite conclusions, especially since differents pecies were probably present. Nevertheless, useful deductions may be made from the findings.

2. The presence of males shows that mosquito breeding occurs near the huts.

3. It is of interest that the sex ratio of mosquitoes is usually 1:1. Unsprayed and BHC-sprayed huts in the Transvaal had an anopheline sex ratio of 1 male:3·9 females, and 1 male:2·4 females respectively;[2] while in the present investigation the culicine sex ratio was 1 male : 4·9 females.

4. From the relatively great scarcity of fully gravid culicines we may conclude that physiological resistance to BHC was absent in culicines during the present investigation, since they were apparently killed by the BHC before becoming fully gravid.

5. The 4 fully gravid females were found just before the second round of residual spraying, i.e. 3 months after the initial spraying. This suggests that BHC remains effective for 3 months.

It must be mentioned, however, that *gambiae* gravidity data revealed that BHC remained effective for 6 months in Bantu huts in the Transvaal, since the first fully gravid *gambiae* females were only found during the 7th month after residual spraying.[1] Exceptions to this were encountered as follows:

(*a*) During the first month after spraying, 1 fully gravid *gambiae* was recovered from blankets hanging in the middle of a very small Bantu hut on the banks of the Limpopo, after it had been unoccupied for 1 week.

(*b*) Fully gravid *gambiae* were found within 7 weeks after spraying of huts with walls made solely from reeds.

6. In this investigation the ratio of unfed: fed and halfgravid females was 1:2·02. With anophelines in the Transvaal, this ratio was 1:0·8. This comparison suggests that the culicines were less susceptible to BHC than anophelines.

7. In research the old tenet that, unless one goes beyond the facts one never gets as far as the facts, applies. The discussion may therefore proceed as follows:

In unsprayed huts in the Transvaal it was originally discovered that *gambiae* matures her eggs indoors. In unsprayed earthen holes we found 80 unfed and 179 fed and gravid *gambiae*. We reasoned that if the female feeds and matures her eggs in 12 hours, gravidity would proceed thus: when gravidity is recorded between 10 a.m. and 4 p.m. we will find — first morning at 10 a.m. 1 unfed female; that night she takes a blood meal at 6 p.m.; on the second morning at 6 a.m. she oviposits, and is again found as unfed at 10 a.m. If she therefore completes oogenesis in 12 hours, she will not be found as fed, or gravid. Similarly if she completes oogenesis in 24 hours, it was calculated that the ratio of unfed: fed and gravid females will be 1:1, but our ratio was actually 80:179 so that she must have matured her eggs in 2·2375 days.[2] This is a good correlation for data determined by other workers on *gambiae*.

In unsprayed huts we found 13 unfed and 130 fed, half-gravid and fully gravid *gambiae*. We reasoned that *gambiae* first rests in the hut before feeding, otherwise we would never have found unfed females, and we calculated that the female rests indoors for $\frac{2{\cdot}2375\times24\times13}{143}$ hours = 4·8818 hours before taking a blood meal. This figure is useful since it is supported by Smith,[3] who determined that 'the peak of fed *gambiae* occurs about 4 hours after the peak of the unfed, suggesting that there is a delay of about 4 hours between arrival at the enclosure wall and feeding'.

If, for the sake of analysis, we further assume that the present culicines behaved like *gambiae* in the Transvaal, we may tentatively conclude that a female becomes half gravid in 1·222 days.[2] Further, if we also assume that the BHC did not kill the culicines, then we would have $\frac{71^{*}}{13}\times107 = 584{\cdot}4$ fed and half-gravid in BHC huts.

We may therefore deduce that BHC might have 'knocked out' the culicine females in $\frac{1{\cdot}222\times24\times216}{584{\cdot}4}$ hours = 10·84 hours. This also indicates that the culicines were less susceptible than *gambiae* to BHC.

SUMMARY

The simplified gravidity technique for determining physiological resistance to BHC,[1] was used in 3 African townships where huts were sprayed with this residual insecticide. The gravidity analysis suggests that BHC remained effective for 3 months, and that resistance was absent. Comparisons indicate that culicines are less susceptible than anophelines to BHC. Using Transvaal data,[2] it was calculated that female culicines were 'knocked out' in 10·84 hours.

I thank Dr. F. A. Donnolly and Mr. R. A. Mansfield for their kind cooperation and continued interest in this simplified gravidity technique.

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(*This is from Steyn et al;[5] 13 unfed and 71 fed and half-gravid *gambiae* females from unsprayed huts.)

ACCIDENTAL INJURIES *

A. D. SMIT, F.R.C.S. (EDIN.), *Orthopaedic Surgeon, East London*

Civilized man reacts to injury and disease in two totally different ways, and, seeing that itis on this basis that present- day medical services have been developed, it is worth while to comment briefly on these reactions and their origins.

There is a deep inherent fear of disease that was born in primitive times when man, baffled by what at that time was an insoluble mystery, full of evil consequences, explained disease on the basis of 'evil spirits', and in doing so frightened the wits out of himself for all time. It is the response to this age-old fear and challenge that has, so far, brought forth the best and most imaginative in medicine.

Physical injury, on the other hand, has for the most part had seemingly obvious causes, and there was no need for our primitive forbears to invent 'spirits' to explain these causes. They were part of their everyday life. They lived with them, joked about them and avoided them when they could. They did not worry unduly about them, and nor do we.

Trauma

In fact, it is only in times of war, and in recent wars at that, that trauma has been faced up to with any spirit of realism. In peace our attitude towards trauma is quite irrational. It is not based on fact, and the system that we have for dealing with it is hopelessly inadequate.

Let me illustrate my point: If an epidemic broke out in Britain tomorrow that killed 45 people a day and seriously disabled 100 more, whatever facilities were needed would be made available, and research into the aetiology, prophylaxis and treatment of the disease would forge ahead. A positive, aggressive, concerted effort would be made to bring it under control. Yet, in Britain today accidents cause just this degree of mortality and morbidity without much being done about it.

The following facts and figures show how serious the position

* From an address to a meeting of the Border Branch of the Medical Association, September 1960.

is in Britain: No less than 80% of the deaths in children under the age of 18 are due to accidents, while, as far as adults are concerned, a workman is in more danger of death and disablement from injury than from disease during his working life. The cost of injuries resulting from traffic accidents in 1955 was £172 million.

In Britain, at the present time, there is a move afoot to bring the situation under control, but it is not receiving much sympathetic attention from authority. The truth is that accidents are as much a part of our lives as sin. They are both a necessary consequence of being us, and we regard them both with the same casual and rather familiar contempt. We joke about them and poke fun at their victims, but it is difficult to jog us into doing anything serious about them.

It is imperative that we shake off the inhibitions of our traditional inherited attitude and carry out a great deal of research into the problem of preventing accidents and healing accidental injuries.

Research

Research of this nature will be difficult, because we will not be dealing with unicellular organisms that, for the most part, live by simple rules, but with *Homo sapiens*, human beings, with their incredible capacity for being unpredictable and stupid.

Until research and legislation bring the vaulting catastrophe of traumatic injuries under control, we, on our side, have the task of reducing to a minimum the tragic effects of trauma. The main question here is: 'Are we fulfilling our task adequately?' The answer is an unequivocal 'No'.

Treatment

I will not attempt to examine all the angles of treatment. The primary treatment is the most important single step in the rehabilitation of the injured, and the measure of its efficiency is a useful index of the efficiency of the rest of the treatment. How good is this primary treatment?

Compound fractures of the tibia are the commonest of severe accidental injuries and the manner in which they are treated gives a good indication of the efficiency of our primary treatment. Here, briefly, is how an average patient with a compound tibial fracture, sustained in a motor accident, is treated:

He is taken to hospital, often with an inadequate splint on his injured leg. In the casualty ward, the stretcher on which he was conveyed and his splint are taken away, and he is placed on a hospital trolly. Frequently, he receives a dressing and another inadequate splint. He is then trundled off to the X-ray department, where the radiographer, who is usually a perfectionist, prefers to work on the X-ray table. The patient, therefore, has to endure 2 moves.

Now he is taken to the ward, where he is moved onto his bed. His clothes are dirty and he is dirty, so the patient is washed and undressed, and cleanliness may literally bring him into close proximity with Godliness.

The surgeon now sees him and gives him some resuscitation. Then he is shifted onto a trolly, taken to the theatre, put on the operating table, and his fractured leg is attended to. Finally, he is put back on a trolly, returned to the ward and put to bed. With inadequate splintage he has been moved 7 times. In all, he has had 9 moves. He has been undressed and washed, and a great deal of precious time has been wasted.

It is obvious that there is little appreciation of the extent of unnecessary human suffering involved, nor is there an appreciation of the deleterious effects of this system.

In mythical times, a highway robber by the name of Procrustes used to offer weary and footsore travellers a bed, with a great show of hospitality. Once in the bed, he forced them to fit it by violently stretching them out, or trimming them down to size with his sword and, in not-so-mythical times, this is the principle we adopt in treating injured cases. We make them fit the system we provide.

Our traditional hereditary attitude is such that we accept all this with complacency and, instead of being shocked, we leave the system to shock the patient.

A good system demands that we reduce preliminaries to an absolute minimum, and that we give adequate definitive treatment.

At the roadside the patient should have his leg adequately dressed and splinted and he should then be placed on a stretcher. He should retain both stretcher and splints until he receives definitive treatment. In the casualty department he should be assessed and X-ray investigation and resuscitation should be undertaken on the spot.

I wish to stress that there is absolutely no need for a patient to be moved from pillar to post. There is no need for him to have his clothes removed. There is no need for him to be washed. These things have no merit in them and there is no shred of evidence to prove that they are of any benefit to the injured individual. They increase his injury and aggravate shock. They are part of the 'Procrustean bed', into which the patient is made to fit.

The municipalities run the ambulance services, and the Provincial authorities run the hospital services. Therefore, any traumatic service which wishes to ensure that a patient remains on a municipal stretcher and wears a municipal splint until definitive treatment takes place, must somehow cut the red tape and try to get the authorities to cooperate. This should be relatively easy, and I am sure that a pool of municipal equipment could be established at hospitals. Then the ambulance driver, handing over equipment, would be able to draw on the pool to replace what he has handed in. For the rest, the simplified system of admission referred to previously should be adopted.

It must be stressed, however, that if the best results are to be achieved the convenience of those who attend the patient can only be allowed a narrow margin of consideration.

Once in the operating theatre, certain basic principles are essential, and in order to ensure that they are observed a systematic method of dealing with cases there is essential; especially when compound fractures are involved. Their serious nature demands standardized treatment of the highest order, if the results are to be satisfactory. Apart from compound fractures, all types of limb injuries are the commonest traumatic injuries treated in hospitals. From my experience their treatment, too, can largely be standardized.

We can only assess results when standardization is achieved, and it is only under a properly controlled system that we can hope to train nursing staff and the rising generation of surgeons. The primary treatment that is given to these injuries is by far the most important step along the road to rehabilitation.

It is time that we make certain that this primary treatment is good, and that all treatment following it comes up to the same standard, so that the primary treatment can have its fulfilment, with the restoration of the individual to his place in society with a minimum of hardship and residual disability.

UNIVERSITEITSNUUS : UNIVERSITY NEWS

VYFDE AKADEMIESE JAARDAG VAN DIE FAKULTEIT VAN GENEESKUNDE VAN DIE UNIVERSITEIT VAN STELLENBOSCH EN DIE KARL BREMER-HOSPITAAL

Die Akademiese Jaardag word vanjaar gehou op Woensdag en Donderdag, 9 en 10 Augustus 1961. Die datums is vervroeg vanweë die Mediese Kongres wat vanjaar in September in Kaapstad plaasvind. Die dinee-dans sal gehou word op Vrydag 11 Augustus. Die program neem 'n aanvang om 2 nm. op Woensdag en word Donderdag voortgesit. Finale programaankondigings sal eersdaags in hierdie *Tydskrif* verskyn.

Geneeshere en ander belangstellendes word hartlik uitgenooi en word versoek om hierdie datums oop te hou. Wetenskaplike en farmaseutiese uitstallings, asook 'n aantal wetenskaplike filmvertonings sal, soos in die verlede, gehou word. Middagete en verversings word verskaf.

FIFTH ACADEMIC STAFF DAY OF THE UNIVERSITY OF STELLENBOSCH FACULTY OF MEDICINE AND THE KARL BREMER HOSPITAL

The annual Academic Staff Day will be held on Wednesday and Thursday, 9 and 10 August 1961. This function will take place earlier than usual this year in view of the Medical Congress which will be held in Cape Town in September. The dinner-dance will be held on Friday 11 August. The programme starts at 2 p.m. on Wednesday and will be continued on Thursday. Final programme announcements will appear in this *Journal* shortly.

Medical practitioners and all others interested are heartily invited to attend and are requested to keep these dates free. Scientific and pharmaceutical exhibitions will be held, as usual, and a number of scientific films will be shown. Lunch and refreshments will be served.

HUNTINGTON SE CHOREA : HUNTINGTON'S CHOREA

'n Suid-Afrikaanse navorser wil graag al die bekende gevalle van Huntington se chorea in hierdie land naspoor. Om die ondersoek te vergemaklik, word alle dokters wat van pasiënte met die siekte weet, versoek om die informasie aan die Redakteur, *Suid-Afrikaanse Tydskrif vir Geneeskunde*, Posbus 643, Kaapstad, te stuur. Hierdie informasie sal dan aan die navorser wat die ondersoek onderneem, gestuur word, en hy sal dan self nadere inligting oor die pasiënte inwin by die persone wat die informasie verstrek het.

A South African research worker is anxious to trace all the known cases of Huntington's chorea in this country. In order to facilitate this survey, all doctors who know of patients with this disease are requested to inform the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town, and the information so obtained will be passed on to the investigator conducting this survey. He will then contact the informant and ask for details about the patient.

DIE LIEFDADIGHEIDSFONDS : THE BENEVOLENT FUND

The following donations during May 1961 are gratefully acknowledged:

Met dank word die volgende skenkings gedurende die maand Mei 1961 erken:

Votive cards in Memory of: Geloftekaarte ter Nagedagtenis aan:

Dr. C. K. O'Malley *by* Dr. J. J. Jacobson; Dr. P. Saling *by* Dr. L. J. Jacobson and Dr. A. J. S. L. Boyde; Mrs. Wynne Appleton *by* Dr. and Mrs. E. Malherbe; Dr. D. A. S. Sichel *by* Mr. V. N. Larsen and Dr. A. A. Cilliers; Mrs. Kuhlmann's mother *by* Northern Transvaal Branch (M.A.S.A.); Mrs. Patter, mother of Dr. D. P. Patter, *by* the Superintendent and Staff of King Edward VIII Hospital; Mr. F. Emerton *by* Dr. R. B. Peckham; Mother Madeline *by* Dr. A. H. Baxter; en Mevr. P. Loubser *deur* Mevr. J. S. du Toit.

Total Received from Votive Cards: / *Totaal Ontvang van Geloftekaarte:* R42.05.

Services Rendered to: Dienste Gelewer aan:

Dr. Louis B. Bok *by* Dr. Leo Mirkin.

Dr. D. F. Fismer *by* Drs. N. M. L. Lund, H. C. Brayshaw, A. J. Pitcher, J. Borrowdale and D. Cowie.

The late two children of Dr. P. Doctor *by* Numerous colleagues in Johannesburg, Port Elizabeth and Uitenhage.

Wife of Prof. J. G. A. Davel *by* Members of the Profession in Pretoria.

Dr. M. Brien *by* Drs. B. Murless, D. Brink and H. M. Curwen.

Total Received from Services Rendered: / *Totaal Ontvang van Dienste Gelewer:* R295.61.

Donations: Skenkings:

Drs. Harries, Kuschke, Hofmeyer, D. T. Trotman-Pitt, C. E. Murray, R. W. Nash, R. R. Richards, H. S. Gear, C. P. Seymour, J. J. Brummer and B. A. Michaelides.

	R
Dr. J. J. W. van Zyl	9.75
Dr. G. J. Cillié	6.60

Total Received from Donations: / *Totaal Ontvang van Skenkings:* R27.20.

Grand Total: Groot Totaal: R364.86.

IN DIE VERBYGAAN : PASSING EVENTS

Messrs. Winthrop Laboratories (S.A.) (Pty.) Limited, have made available a small collection of medical films which would be of interest to members of the medical profession. This firm have advised that the showing of these films would be willingly organized if required. Further information may be obtained from Messrs. Winthrop Laboratories (S.A.) (Pty.) Ltd., P.O. Box 8900, Johannesburg.

* * *

The Seventh International Congress of Internal Medicine is being held in Munich from 5 to 8 September 1962. The main subjects it is proposed to discuss are: (1) Immune phenomena in the pathogenesis of internal diseases; (2) The influence of the milieu in some diseases (rheumatism, hypertension, diabetes mellitus, arteriosclerosis, myocardial infarction); and (3) Shock and collapse in internal diseases.

Time will also be allowed for papers on other subjects. Those physicians who wish to read papers or to take part in this congress are requested to communicate with Dr. Pericles Menof, 607-608 Medical Centre, Jeppe Street, Johannesburg.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 14 June at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. P. Helman will speak on 'Venous ulcers of the leg'. All members of the Medical Association are welcome to attend this lecture.

* * *

Dr. S. Burgin, M.B., B. Ch. (Rand), F.C.O.G. (S.A.), M.R.C.O.G., has commenced practice as an obstetrician and gynaecologist at 120 Vannel Building, Church Street, Klerksdorp. Telephones: Rooms 24697, residence 23523.

Dr. S. Burgin, M.B., B.Ch. (Rand), L.K.V.G. (S.A.), M.R.C.O.G., het begin praktiseer as verloskundige en ginekoloog te Vannelgebou 120, Kerkstraat, Klerksdorp. Telefone: Spreekkamers 24697, woning 23523.

Dr. B. T. Bernstein, of Johannesburg, has returned from a visit to otological clinics in Israel, London, and the United States of America.

* * *

Dr. C. W. Coplans, of Cape Town, has left on a short visit to the United Kingdom. He will be away for about 1 month and will visit physical medicine centres in that country.

* * *

Smith and Nephew Associated Companies Ltd. Awards for Post-graduate Research. Six new fellowships to be awarded to overseas doctors, have been established by one of Britain's leading medical houses, Smith & Nephew Associated Companies Limited. The Fellowships, worth £1,200 (R2,400) a year each and tenable only in the United Kingdom, will go to medical graduates selected from British Commonwealth and developing countries and will be available in any branch of surgery or associated research.

In establishing the awards so that outstanding graduates can further their education to the benefit of their individual countries, Smith & Nephew have stipulated that candidates must agree to return home to practice within a year of the completion of their studies. Fellows may be of either sex and must hold a medical qualification registrable in Britain and have had at least two years' general clinical experience since registration.

Known throughout the world for its medical and surgical products, Smith & Nephew have a long tradition of active, though unobtrusive, charitable help to the medical world, particularly in underdeveloped territories. For many years, through the initiative of the Group's late President, Mr. H. N. Smith, they have been closely associated with hospitals in Swaziland, missionary and leper hospitals in Africa, Korea and India, and have collaborated with many overseas governments in extensive field trials into the treatment of tuberculosis.

Application forms, which must be completed and returned to England by 31 July 1961, can be obtained from the Secretary, Smith & Nephew Associated Companies Ltd., 2 Temple Place, Victoria Embankment, London, W.C. 2.

The 'Lindsay Sandes' Wing of the Victoria Hospital, Wynberg, was opened on Friday 26 May 1961 by Mr. Lionel Murray, M.E.C. A large number of practitioners and other interested persons were present.

In addressing those present, Mr. Murray said that this new wing was yet another tangible example of the tremendous growth in hospital services provided by the Cape Provincial Administration. The wing, which has 80 beds, was named after Dr. Thomas Lindsay Sandes, honorary surgeon at the hospital for 30 years and senior lecturer in surgery at the University of Cape Town. Dr. Sandes was also associated with the other major hospitals in the Cape and was awarded the gold medal of the Medical Association and the Order of the British Empire (Military Division) for his services.

'This building will perpetually commemorate the name of a distinguished surgeon, a humanitarian and one of the most lovable members who have graced the medical profession of this city and the country', Mr. Murray said.

The wing provides for storage in the basement, space for 22 infants and children on the ground floor, and 30 beds for White men and 30 for White women on the first and second floors respectively.

At the conclusion of his address, Mr. Murray unveiled a memorial plaque of the late Mr. Lindsay Sandes.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

PERIACTIN

Merck Sharp & Dohme announce the introduction of Periactin and supply the following information:

Periactin combines antihistaminic and antiserotonin properties. Periactin is highly potent in both phases of its anti-allergic activity, being comparable to the most active antihistaminic and antiserotonin substances known. Its concentration in the skin following oral dosage appears to be superior to that of other antihistamines. Moreover, in animal studies it has equalled or surpassed the antiserotonin effects of lysergic acid diethylamide (LDS), 1-benzyl-2-methyl-5-methoxytryptamine (BAS), and 1-benzyl-2-methyl-5-hydroxytryptamine (BMS).

Periactin is well tolerated and has an unusually wide range of activity in the treatment of acute and chronic allergies, and is recommended in such conditions as: hay fever and other seasonal rhinitis, perennial allergic (vasomotor) rhinitis, urticaria, angioneurotic oedema (Quincke's disease), atopic dermatitis, such as eczema (eczematoid dermatitis and both localized and disseminated neurodermatitis), drug and serum reactions, contact dermatitis (including dermatitis venenata, e.g. due to plants), neurotic excoriations, insect bites and stings, sunburn, and pruritus, even when not necessarily associated with allergic phenomena.

Side-effects

Drowsiness has been observed to occur in some patients. This side-effect is often desirable in patients with dermatitis and pruritus, since it tends to raise the threshold of perception, and may decrease emotional tension and the tendency to scratch caused by the disease.

Some dizziness, nausea and dry mouth have been reported in low incidence.

Patients who become drowsy on Periactin should be cautioned against driving a car or operating machinery or appliances requiring alert attention.

Dosage

Dosage must be individualized. The therapeutic range is 4 - 20 mg. a day, with the majority of patients requiring 12 - 16 mg. a day. An occasional patient may require as much as 32 mg. a day for adequate relief. It is suggested that dosage be initiated with 4 mg. 3 or 4 times a day and adjusted according to the weight and response of the patient.

Paediatric Dosage

The dosage for children between the ages of 2 and 14 years is 6 - 16 mg. a day, depending upon the weight and response of the patient. The initial dosage is usually 2 mg. 3 or 4 times a day.

Since the effect of a single dose usually lasts 4 — 6 hours, the daily requirements should be given in divided doses 3 or 4 times a day or as often as necessary to provide continuous relief.

Further information is available on request from Merck Sharp & Dohme (Pty.) Ltd., P.O. Box 7748, Johannesburg.

BOEKBESPREKINGS : BOOK REVIEWS

OBSTETRICS AND GYNAECOLOGY

Clinical Obstetrics and Gynecology. Vol. 3, No. 1. Obstetric Emergencies edited by Martin L. Stone, M.D. Pediatric Gynecology edited by John W. Huffman, M.D. Pp. 264. Illustrations. Subscription to series: $18.00 a year. New York: Paul B. Hoeber, Inc. 1960.

During the past 20 years the main object of the obstetrician has been to reduce the infant and maternal mortality. Antibiotics and better aseptic techniques have mainly been responsible for a marked drop in infant and maternal mortality, but in spite of all our modern methods, there are still a number of preventable maternal deaths. These maternal deaths are mainly from the so-called obstetric emergencies; hence a detailed knowledge of this branch of obstetrics is absolutely essential to all practising obstetrics. The first part of this volume contains a concise account of all these emergencies as well as a detailed account of their treatment. Some of the opinions expressed are perhaps controversial, but the whole subject is so well discussed that it can be highly recommended to medical students, general practitioners, and consultants.

The second part of this volume on paediatric gynaecology is of particular interest since there are very few accounts of this subject as a whole. The infrequency with which the practising gynaecologist sees gynaecological paediatric problems, sometimes makes the diagnosis difficult and the management uncertain. The authors have thoroughly realized this and have thus given a compact account of the subject without sacrificing any detail—so that it can also be used as a reference book for the busy practitioner.

Finally, I can only congratulate the authors on this valuable addition to our gynaecological library. R.W.A.N.

ACTION OF INSULIN

The Mechanism of Action of Insulin. A Symposium organized by the British Insulin Manufacturers. Consulting Editor: F. G. Young, M.A., D.Sc., F.R.S. Editors for the British Insulin Manufacturers: W. A. Broom, B.Sc., F.R.I.C. and F. W. Wolff, M.D. Pp. xvi+320. Illustrated. R3.25. Oxford: Blackwell Scientific Publications. 1960.

This book strikes me (who also attended the symposium which it reports) as fascinating, chiefly because it indicates to some extent the vast amount of work which has been and is being done on insulin, and how very little is yet known of its precise modes of action. It is difficult to single out individual contributions. We have, among others, Nicol discussing the different chemical varieties of insulin; Chain (a co-discoverer of penicillin and Nobel-prize winner) giving his entirely revolutionary ideas on the action of insulin, backed by some fantastic machines for 'quantitative bidimensional radio-chromatography'; de Duve upholding the view that insulin *does* act directly on the liver; several of the insulin bio-assay experts discussing their methods, results and the insulin-antagonists; Butterfield's fascinating red-herring which started with burns; and Levine's charming summary of present views on the mode of action of the sulphonylureas. Finally we are allowed a little clinical excursion into prediabetes.

Anyone interested in the basic disorder in diabetes or in carbohydrate metabolism should read this volume. The symposium as a whole was a fine illustration of cooperation between four important British drug firms (comprising the British Insulin Manufacturers) for the benefit of medical science. Similar efforts would be welcomed in this country. W.P.U.J.

THORACOTOMY

Techniques of Thoracotomy. By B. T. le Roux, M.B., Ch.B., F.R.C.S.E. Pp. xi+94. 65 illustrations. R5.50. Edinburgh and London: E. & S. Livingstone Ltd. 1961.

Surgical technique is largely an individual expression of the art which surgeons practice, and, as such, it is controversial. The techniques of thoracotomy are no exception. Whereas most thoracic surgeons will differ from Mr. le Roux on minor details, no one can fail to benefit by reading his detailed accounts of how to gain access to the thorax and how to close a chest. For the surgical apprentice and the general surgeon who sometimes ventures into the chest, this monograph should provide a valuable guide.

Important principles are briefly referred to in the introduction. The author's approach and closure for all the usual intrathoracic and thoraco-abdominal procedures (practised by the Regional Thoracic Unit, Edinburgh) are described in detail, from the induction of anaesthesia until the patient leaves the theatre.

Production is excellent and the numerous colour photographs more than compensate for the price of this small monograph. It was a pleasure to read an account based on personal experience, and refreshing to find a text unmarred by references.

G.S.M.B.

YEAR BOOK OF ORTHOPAEDIC SURGERY

The Year Book of Orthopedics and Traumatic Surgery, 1959-1960. Edited by R. K. Ghormley, M.D. and H. H. Young, M.D. With a section on Plastic Surgery, edited by N. Owens, M.D., F.A.C.S., F.I.C.S. Pp. 448. 217 illustrations. $8.00. Chicago: The Year Book Publishers, Inc. 1960.

In spite of the death of Dr. Ralph K. Ghormley, this year's Year Book is well up to the standard of its predecessors.

As evidenced by the various papers, there is an increasing realization of the importance of a basic training in peripheral vascular surgery for those surgeons who are dealing with trauma generally and fractures in particular. So much can be done now by various techniques, that it is certain that more limbs, or portions of limbs, will be saved as a result of this knowledge.

The use of polyurethan polymer (Ostamer) is reported in this issue. As yet finality has not been reached as to the desirability or otherwise of its properties in bone surgery. This controversial substance has also been the subject of an editorial in the *Journal of Bone and Joint Surgery*.

It is a particular pleasure to note in this issue abstracts of two papers which originally appeared in the *South African Medical Journal*.

This year's edition is strongly recommended to those who wish to 'keep up with their reading'.

A.S.

PAEDIATRICS

Advances in Pediatrics. Vol. XI. Edited by S. Z. Levine. Pp. 285. Illustrated. $9.00. Chicago: Year Book Publishers, Inc. 1960.

This excellent volume presents six up-to-date monographs each with its own extensive bibliography. The topics chosen fall into two groups, one intended for the general practitioner and the other for the paediatrician.

The practitioner will enjoy an easy-to-read article on respiration and respiratory diseases of the newborn, and will learn a great deal about hyaline membrane disease, meconium aspiration, and other syndromes which cause respiratory distress. He will be pleased by a practical article on hormone therapy in rheumatic fever, in which the diagnosis of carditis is detailed and the value of short-term steroid therapy is stressed.

In a chapter on diarrhoea and dehydration, the methods used in a Chilean outpatient department are outlined—a useful article since our conditions are so very similar. An article on allergy teaches an approach to that common problem, the allergic child.

Hypo- and hypernatraemia and the neurologic regulation of sodium concentration are discussed under the formidable title 'Clinical and experimental interrelationship of sodium and the central nervous system'. To complete this satisfying volume there is a very interesting chapter describing the clinical syndromes in which the urinary excretion of amino acids is abnormal.

All in all a very valuable book. L.S.

GENERAL ANAESTHESIA FOR NEUROSURGERY

A Practice of General Anaesthesia for Neurosurgery. By R. I. W Ballantine, M.R.C.S., L.R.C.P., D.A., F.F.A.R.C.S. Pp viii+152. 68 illustrations. R2.75 net. London: J. & A. Churchill Ltd. 1960.

In line with the growing tendency to produce monographs on specialized aspects of anaesthesia, this book is a most welcome addition. The authors describe the various problems in the field of anaesthesia for neurosurgery, and then, systematically, the means of combating them. Posture-induced hypotension and hypothermia are adequately described. There is a useful section on head injuries, and, finally, a chapter on pre- and postoperative care.

Possibly the most important feature of this book is the description of the various mechanical and technical difficulties which may arise during the course of long operations. All these problems have been described in the literature, but it is difficult for those infrequently confronted by neurosurgical problems to obtain an adequate, complete picture.

For the anaesthetist who finds general anaesthesia for neurosurgery difficult and demanding, this book will be helpful, and can be warmly recommended. P.S.J.

PSYCHOTHERAPY

Psychotherapists in Action. By Hans H. Strupp. Pp. xiv+338. $8.75. New York and London: Grune & Stratton, Inc. 1960.

The active processes which wreak change in the patient as a result of psychotherapy are not completely known. Certainly a major factor lies in the personality of the therapist and in his relationship with the patient, but since therapists are not standard units there is an enormous amount of variation of this factor which extends also to their clinical observations, judgements, and therapeutic operations. All these are extremely difficult to evaluate because of the highly subjective nature of psychotherapy and the many intangible human elements that are involved.

The author, by means of an ingenious technique, has managed to invade this private realm to give us a fascinating picture of what goes on in the psychiatrist's mind as he works, and the transactions that take place between him and his patient. A standard film sequence of an unrehearsed psychotherapeutic interview was shown to a series of 237 psychotherapists ranging from real experts to veriest newcomers, and belonging to all schools of current psychiatric thought. They were then questioned according to a detailed protocol for their reactions to the screened situation—what they thought of the therapist's technique, the patient's reactions, how they would have handled it, the prognosis, psychodynamics, etc. The results are then analysed in a workmanlike statistical fashion, and the result is an absorbing and enlightening survey of contemporary psychotherapy. The final section of the book is devoted to an analysis of psychotherapeutic techniques and a general discussion of the factors and problems involved in research on psychotherapy.

All in all this is a most rewarding and unique book, and all psychiatrists and psychologists who take their psychotherapy seriously will gain much from the rich fund of material it contains.

L.S.G.

OPHTHALMOLOGY

Synopsis of Ophthalmology. By William H. Havener, B.A., M.D., M.S. (Ophtha.). Pp. 288. 189 illustrations. South African price R5.75. St. Louis: C. V. Mosby Co. 1960.

Another of the synopsis series, this volume is particularly useful for undergraduate reference in a speciality often neglected for lack of short, concise teaching.

Attractively presented and well indexed, this little book deals comprehensively and concisely with all the common, and many rather less common ophthalmic disorders.

The chapter on medical ophthalmology and that on treatment, discussed by means of illustrative cases, are particularly useful.

This book is recommended to all medical students and to general practitioners who wish to gain some knowledge of ophthalmology without having to wade through the larger reference books. P.A.T.

THE OLDER PATIENT

The Older Patient. By 21 authors. Edited by Wingate M. Johnson, M.D. Pp. 589+xiv. Illustrated. $14.50. New York: Paul B. Hoeber, Inc. 1960.

This book is well planned to cover the field of geriatric practice. Each chapter has been carefully selected, in order to bring the particular problems of the older patient into prominence.

The importance of anatomic changes which occur with age are stressed. The particular diagnostic aspects of disease in this age group are described and there are excellent sections devoted to such matters as nutrition, relief of pain, and surgical procedures.

Of importance too is the emphasis placed on psychological factors, and there is a special chapter on mental disorders of the aging. The various therapeutic approaches are adequately set out.

This volume should prove to be a very practical guide to the diagnosis and management of the older patient—a field of medical practice which is so rapidly growing in importance. A.J.B.

PAEDIATRICS FOR NURSES

A Handbook of Paediatrics for Nurses in General Training. 2nd Edition. By Q. M. Jackson, D.N. (Lond.). Pp. viii+105. R1.25 net. London: H. K. Lewis & Co. Ltd. 1960.

This is a noble effort to give to the student-nurse who is in general training some knowledge of paediatrics. To do that in 100 pages of a small book with relatively large print is a formidable task and criticism must be tempered by the limitation of space. Quite a lot of medical information could have been omitted and more nursing detail put in, e.g. for gastric lavage, how far is the tube to be passed and how much fluid used at each filling? Some archaic and unnecessary manoeuvres should by this time have been recognized as such, e.g. strapping a coin on an umbilical hernia and the necessity for interruption of a baby's feed to get up wind. History-taking and the normal haematological variations are not needed for nursing, but at least there can be no complaint that the buyers will not get value for their money. F.J.F.

BRIEWERUBRIEK : CORRESPONDENCE

PROTAGORAS, DR. JONES, AND LARYNGEAL INTUBATION

To the Editor: The Victorian's unquestioning faith in his own scepticism was tremendous, something Dr. Jones rediscovers in the *Journal*[1] when (to him) 'it seems odd that Man (in this case an anaesthetist) should consider himself better able than Nature (or God) to sustain natural functions in his fellowmen'. Now we could go to the other extreme of misguided humanism that sees man as the source and centre of all values, religion among them, and say with the ancient Protagoras of Abdera: 'Man is the measure of all things'. Thus we would be more in harmony with the New World *Man versus God* theme, but we shall be just as remote and irrelevant, just as highly literately ignorant and stupid about laryngeal intubation as we believe Dr. Jones is in his unambiguous indictment of responsible modern anaesthetic practice. Exactly like those who, for various motives of expediency and insecurity, refuse to use clearly superior new drugs, Dr. Jones condemns intubation on the slenderest possible grounds, and offers no alternative other than a return to the nurse-anaesthetist era. That surely is the issue to be faced; one may agree or disagree, but let us not take refuge in vague metaphysical inanities! Perhaps Dr. Jones wanted to suggest that intubation is wrong in the absence of an indication. That is good advice. But as his article stands, there is the comparable view of an Edinburgh anaesthetist, T. D. Luke, to which one should draw his attention (written in 1908):[2]

'Some surgeons say that there is one anaesthetic and that is chloroform. Chloroform may be given anyhow, anywhere, by anyone. If the patient shows signs of impending dissolution he is said to take the chloroform badly. If he does not, he takes it well. If he dies it is an act of God. An anaesthetist is defined as a latter-day nuisance, of no use to the surgeon and a curse to the G.P. There is no more need for the anaesthetist than for a hypodermic injectionist or other poisonist'.

J. W. Mostert

Department of Anaesthetics
University of the Witwatersrand
30 May 1961

1. Jones, C. S. (1961): S. Afr. Med. J., 35, 421.
2. Sykes, S. W. (1960): *Essays on the first hundred years of Anaesthesia*, p. 101. Edinburgh: Livingstone.

SHORTAGE OF GENERAL PRACTITIONERS

To the Editor: There is an acute shortage of general practitioners in the Union, just as there are too many specialists.

According to the numbers of medical students in the various years in the medical schools in this country, this shortage will continue for years.

Locums are at a premium now, and are most difficult to obtain.

Before the war a locum was paid £2 2s. a day and all found. If the pound is worth 8s. today, then a remuneration of £5 5s., plus all found, should be ample.

After all, most locums are only qualified a year or two. A fee of 5 guineas a day and all found is now sneered at, and at this figure locums are unobtainable. One has to pay £6 — 7 and all found per day or even more.

These gentlemen are exploiting their own colleagues.

A proposal at a Federal Council meeting a few years ago that the salaries of locums should be fixed at 5 guineas a day, could not find a seconder.

The Central Committee for Contract Practice was instituted to prevent medical aid societies from exploiting the profession, and *vice versa*. This the Central Committee for Contract Practice has done very ably.

No machinery, however, exists for preventing a section of the medical profession from exploiting another section.

Harassed general practitioners, who have not had a holiday for years, are sometimes prepared to pay as much as £10 and all found a day for a locum—a good deal more than they themselves actually earn.

Some of the gentlemen even request that you also put up their wives, and sometimes the odd child and even a dog. Their attitude is truly amazing.

Very few have bags containing sphygmomanometers and auroscopes, some even arrive without a thermometer or stethescope, impossible as it may seem.

An apprentice on a mine has to buy some tools out of his meagre salary; a carpenter, fitter or turner has to have about £60 worth of tools when he takes on a job anywhere, yet our friends, the locums, with salaries of £200 and all found per month, do not even have the barest essentials to carry out the duties of their profession.

It is high time the Federal Council of the Medical Association took some action to prevent a section of the profession from exploiting another section.

Takhaar

23 Mei 1961

NEW SICK-FUND FEES FOR GENERAL PRACTITIONERS

To the Editor: As I am a doctor practising outside a municipal area, I would like to record my objection to the fact that our fees for visits to patients' homes is to remain at 25 cents less than the fees of our colleagues who live within the municipal boundaries.

I base my objection on the following grounds: A record of mileage and time expended on my last 100 calls show that my average mileage per call, and time spent on travelling, was 3·2 miles, and 9 minutes respectively. To receive 25 cents for this service, I think is ludicrous. A fee of 50 cents above the consultation fee is surely not unreasonable.

Most patients do come to the surgery, but some prefer that one should call at their homes—to save them the expense of travelling, which is often more than 25 cents, and then still have to wait in the consulting rooms.

A further point, which has been raised before, is that it is not right that patients and doctors living outside municipal areas should subsidize the ordinary travelling expenses of doctors living within municipal boundaries.

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23 May 1961